THE EFFECT OF ACUTE AND CHRONIC INSPIRATORY MUSCLE LOADING UPON ROWING PERFORMANCE

by

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ABSTRACT

The study of exercise physiology involves the integration of the physiology of many systems. The determination of athletic performance is an amalgamation of yet more factors drawn from not only physiology, but also psychology and biomechanics. The subject of this thesis incorporates various aspects of respiratory and exercise physiology (control of breathing, dyspnea, perceived exertion, respiratory mechanics, warm-up, hypoxemia, muscle physiology, etc.) that it would not be appropriate to discuss in a comprehensive manner. Thus, the approach that has been adopted in the introduction is to present only a distillation of the most relevant and contemporary research in these areas, in order to provide the scientific background for the research chapters that follow.

Even though it is traditionally thought that ventilation does not limit exercise performance in the healthy adult, in recent years it has been demonstrated that individuals with a high work capacity may be prone to respiratory limitations. Respiratory limitations may arise in terms of gas exchange, respiratory mechanics, energetics of the respiratory muscles, or because of the development of respiratory muscle fatigue.

During rowing the combination of the entrained breathing pattern, the mechanical limitations of the pulmonary system and the additional static supportive work for the upper body, place high demands upon the respiratory muscles. These demands predispose the respiratory muscles to fatigue despite of the high fitness levels observed in rowers. Due to the various implications that respiratory muscle fatigue can have upon rowing performance, the aim of this thesis will be: a) to investigate the incidence of respiratory muscle fatigue during rowing, b) to reduce respiratory muscle fatigue by means of inspiratory muscle training and a specific respiratory warm-up and c) to evaluate the effect of such interventions upon rowing performance.

Contents

List of abbreviations

Abbreviation	Term
BPNS	Bi-Phrenic nerve stimulation
CO_2	Carbon Dioxide
COPD	Chronic obstructive pulmonary disease
CV	Coefficient of variation
ECG	Electrocardiogram
EEV	End-expiratory volume
EIV	End-inspiratory volume
f_R	Breathing frequency
f_C	Cardiac frequency
FEV_1	Forced expiratory volume in 1 second
FRC	Functional residual capacity
FVC	Forced vital capacity
Hz	Hertz
IMT	Inspiratory muscle training
L_o	Optimal length for muscle force development
$[Lac]_{BL}$	Blood lactate concentration
MLSS	Maximum blood lactate steady state
MVV	Maximum voluntary ventilation
O_2	Oxygen
Pa_{CO_2}	Partial arterial pressure of carbon dioxide
PE_{max}	Maximum expiratory pressure
P_{ETCO_2}	End-tidal Carbon dioxide pressure
P_{ETO_2}	End-tidal Oxygen pressure
PEFR	Peak expiratory flow rate
PIFR	Peak inspiratory flow rate

PImax	Maximum inspiratory pressure
MIP	Maximum inspiratory pressure
P_{pl}	Pleural pressure
$P_{pl\ cap}$	Pleural pressure capacity
RV	Residual volume
Sa_{O_2}	Arterial oxygen saturation
SD	Standard deviation
T_I	Inspiratory duration
T_{TOT}	Total breath duration
VC	Vital Capacity
$\dot{V}_{CO_2}$	Minute Carbon dioxide output
$\dot{V}_E$	Minute ventilation
$\dot{V}_A$	Alveolar ventilation
$\dot{V}_E/\dot{V}_{O_2}$	Ventilatory equivalent for oxygen
V_T	Tidal volume
$\dot{V}_{O_2max}$	Maximum Oxygen uptake
$\dot{V}_{O_2}$	Minute Oxygen uptake

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Publications

Volianitis S, McConnell AK, Koutedakis Y, Jones DA (1999) The influence of prior activity upon inspiratory muscle strength in rowers and non-rowers. *Int J Sports Med* **20**: 542-547.

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Chapter One

Introduction

1-1. Breathing During Rowing

An appreciation of the pattern of breathing adopted in rowing requires an explanation of the total ventilatory response and the factors that influence the depth and frequency of breathing. These factors can be divided into two categories. The first consists of factors that influence the total ventilation and are mainly dependent on metabolism, the gas exchange efficiency, and mechanisms for the control of breathing. The second category consists of mechanical factors that tend to modify the pattern of breathing with certain constrains arising mainly from resistive and elastic forces and the capacity of the respiratory muscles to overcome those impedances.

1-1.1 Factors Contributing to Ventilation

Metabolism

Energy requirements

The majority of rowing competitions are raced over 2000 metres, although during many locally organised Head of the River events rowers may compete over longer distances. Depending on the type of boat, a typical race may require between 220 and 250 strokes to complete, with the forces being exerted on the oar handle for each stroke being the equivalent of 400-500 N. As a consequence rowers require very high muscular strength and power in order to sustain maximal effort during a race which typically lasts between 5.5 to 7.5 minutes.

A study which used actual rowing times (Secher, 1983) calculated the metabolic cost to be about 6.4L O_2/min, assuming a constant mechanical efficiency of 22 percent. Rowing a six-minute "all-out" effort on an ergometer has been used as a simulation of rowing competition (Hagerman, 1971; Secher, 1982). Even though, the ideal method of physiological testing is to measure oxygen consumption on the

water, using a Douglas bag (Jackson & Secher, 1976; Chenier & Leger, 1991) or a modern telemetric oxygen consumption monitor (Kawakami *et al*, 1992), a kinematic comparison of rowing and rowing ergometry (Lamb, 1989) has shown that the dominant movements of the legs and trunk are similar for the two types of activity and ergometry yields very similar aerobic power values to those obtained on the water (Martindale & Robertson, 1984).

During ergometer rowing, Droghetti *et al* (1991) calculated the metabolic cost of a 6 min all-out effort with 33 strokes/min, and an average work output of 410 watts, to be approximately 7.0 L O_2/min.

Oxygen uptake ($\dot{V}_{O_2}$) *requirements*

Over the course of a race, elite rowers have been found to exercise at what may be called "severe steady state", with the majority of work performed at between 95 and 98% of maximal aerobic capacity. In male national level athletes, a $\dot{V}_{O_2}$ of up to 6 L/min and up to 6.5 L/min in elite male athletes can be expected. For female athletes, the corresponding values are about 4 L/min and 4.4 L/min, respectively.

The racing pattern commonly used in rowing is unique. This is depicted by a short sprint of about 40 seconds at the start, requiring a large energy turnover, placing large demands on the anaerobic energy system, and a high stroke cadence of about 40-50 strokes per minute. During the middle part of the race the stroke rate is reduced to approximately 35 strokes per minute before increasing again over the last 500 metres. A physiological explanation for the initial spurt performed is given by Secher *et al* (1982) who suggest that the rate of increase in oxygen uptake at the onset of exercise is highest the greater the work load. Accordingly, the total oxygen uptake and work output during a given period of exercise is larger when an initial spurt is performed than when one attempts to keep exercise at an average intensity throughout a race (Secher *et al*, 1982). Oxygen uptake reaches

its highest value after 90 sec and balances out at a level that shows only a slight decrease during the remaining 4 minutes.

Carbon dioxide ($\dot{V}_{CO_2}$) *Production*

The $\dot{V}_{CO_2}$ is significantly above the $\dot{V}_{O_2}$ from the second minute onwards and that results to a respiratory exchange ratio (RER) which exceeds 1.15. Up to the fourth minute, $\dot{V}_{CO_2}$ increases exponentially and thereafter exhibits a slight, almost linear increase. Towards the end of the maximal test simulating racing conditions, $\dot{V}_{CO_2}$ is more than 0.5L/min higher than the $\dot{V}_{O_2}$ (Hartmann, 1993).

Gas Exchange Efficiency

The alveolar ventilation ($\dot{V}_A$) required depends on the level of the arterial tension of carbon dioxide (Pa_{CO_2}) which is to be regulated. At any set point of Pa_{CO_2} the demands for $\dot{V}_A$ increase as a linear function of $\dot{V}_{CO_2}$. But a lower Pa_{CO_2} set point would require a higher $\dot{V}_A$ for the same $\dot{V}_{CO_2}$. The increase in ventilation needed to reduce Pa_{CO_2} by a particular amount is progressively greater the higher the $\dot{V}_{CO_2}$ (Whipp & Ward, 1998). Therefore elite rowers have to increase their $\dot{V}_A$ appreciably more than moderately fit rowers with lower achievable metabolic rates and related $\dot{V}_{CO_2}$.

The anatomical and alveolar dead space contribute to the differences between minute ventilation ($\dot{V}_E$) and alveolar ventilation. The ratio of dead space to the tidal volume (V_D/V_T) is an index of ventilatory efficiency with respect to gas exchange (Jones, 1991). The high V_T achieved by rowers decreases V_D/V_T to a value as low as 5% despite the actual increase of the dead space during exercise

(Whipp & Ward, 1998). A low V_D/V_T requires less $\dot{V}_E$ to maintain a given level of Pa_{CO_2} as work rate is increased.

Minute Ventilation ($\dot{V}_E$)

The dynamic characteristics of the $\dot{V}_E$ response to a high level of exercise at which a sustained lactic acidocis ensues are complex, non-linear, and often a steady-state is unattainable. For moderate intensity exercise the CO_2 exchange can be considered the dominant determinant of the magnitude of exercise hyperpnea, but during rowing competition the metabolic acidosis leads to additional $\dot{V}_E$ drive that provides respiratory compensation for the acidosis. Additionally, circulating catecholamines, high body temperature and increased blood osmolarity may also contribute to the hyperventilation seen at these high work rates. In addition, increases in plasma potassium have been related to hyperpneic responses during rowing (Newstead, 1990).

Very large respiratory minute volumes are developed during competition, typically greater than 200 L/min and sometimes as high as 250-270 L/min(McKenzie & Rhodes, 1982). Performance capacity is more favourable for rowers with large total lung and vital capacities (Donnelly *et al*, 1991). The importance of initial selection of future rowers is suggested, since intensive training does not increase total lung capacity or vital capacity once adult stature has been attained (Danuser & Buhlman, 1983).

Some reports suggest that in rowers the ventilatory response to exercise is usually characterised by a low ventilatory equivalent ($\dot{V}_E/\dot{V}_{O_2}$)(Mahler, 1991a; 1991b; Secher, 1983) presumably because the rower maintains a cramped body position during the initial or catch phase of the stroke, thus impairing normal excursion of the diaphragm (Cunningham, 1975). However, earlier reports suggest that ventilatory equivalents for rowers equal or exceed those for most other endurance athletes (Hagerman, 1972; 1975; 1975a; 1975b; 1984).

These discrepancies suggest, as argued by Whipp & Ward (1991), that inter- and intra-individual variations in the levels of RER, the set point for Pa_{CO_2}, and the efficiency of the lung (V_D/V_T), can require levels of minute ventilation ($\dot{V}_E$) for a particular work rate that vary as much as four times. They also suggest that $\dot{V}_E$ should be measured directly and not predicted. In this respect we site previous data describing the pattern of the ventilatory response to simulated rowing ergometry (Hartmann, 1993). During the 6 min all-out rowing $\dot{V}_E$ rises exponentially until the third minute. At this point the rate of increase slows down, but nevertheless, it continuous to increase until the end of the test (Hartmann, 1993).

1-1.2 Mechanical Factors: Impedances to Breathing

Determinants of Mechanical Breathing Pattern

The $\dot{V}_E$ and the pattern of breathing are the result of the pattern of respiratory muscle contraction acting on the mechanical properties of the respiratory system. The extent of the tidal volume is determined by the size of the lungs, and both inspiratory and expiratory times are influenced by maximum airflow. The lungs of rowers reflect their large bodies. Vital capacities up to 9.1L have been recorded (Secher, 1983). Maximal V_T during simulated competitive rowing average ~55% of vital capacity (Siegmund *et al*,1999), which is very close to the asymptotic plateau described by Hey (1966).

In the Hey plot, the relationship between V_T and $\dot{V}_E$ is expressed graphically in terms of two straight lines, and it depicts the V_T increasing up to an asymptote of about ~60% of vital capacity, above which point increases in $\dot{V}_E$ are mainly achieved by increases in breathing frequency. Inspiratory flow rate is limited

primarily by the ability to generate inspiratory muscle pressure (P_{mus}). In contrast, expiratory flow rate is limited by airway mechanics, not the ability to generate expiratory pressure. Peak expiratory flow rates reach values of 15 L/sec in elite male competitors, but some of these individuals also show a plateau in expiratory flow rates suggestive of airway collapse (Carles *et al*, 1980; de Swiniarski, 1990; Steinacker *et al*, 1993). The net dynamic pressure resulting from the contraction of all the inspiratory muscles (P_{mus}) can be calculated by the following equation:

$$P_{mus} = P_{el} + P_{res} - P_{pl}$$

where P_{el} is the pressure used to overcome chest wall elastance , P_{res} is the pressure used to overcome resistance and P_{pl} is the pleural pressure (Younes and Kivinen, 1984). P_{pl} has frequently being used as an index of net inspiratory muscle pressure (P_{mus}) by ignoring P_{el} and P_{res}. However, these two components can make up a significant fraction of P_{mus} in cases where compliance of the chest wall is reduced and resistance due to increased flow rates is increased.

A study investigating the influence of heavy rowing work on the lungs of female rowers suggested that female rowers have a higher specific static and dynamic lung compliance and a lower recoil pressure at functional residual capacity as compared with other female athletes or male rowers (Biersteker *et al*, 1986). The physical stress on the thorax during rowing is not alike for women and men. In contrast to men, women have to contract additional muscle groups to stabilise their longer trunk during the drive phase of the stroke (Hoske, 1953). For the same body height, women have a lower fat-free mass and a greater trunk length than men. This makes their trunk less stable as compared with men. The extra trunk length of about 2 cm is located in the abdominal region for reproductive purposes. In activities where the trunk is used as a lever for large forces women have to fixate the diaphragm and the abdominal muscles. Measurements of transpulmonary pressure during rowing have indeed shown an intrathoracic

pressure rise during the pull phase of the rowing stroke in female rowers, but not in male rowers (Biersteker *et al,* 1986).

The reduced lung elastance was interpreted as being responsible for the significant expiratory flow limitation observed in the female subjects of Biersteker *et al,* (1986). A similar reduction in lung elastance, but to a lesser degree, was also present in the male subjects of Steinacker *et al,* (1993). An expiratory flow limitation results in a dynamic hyperinflation of the lungs which a) increases the elastic work and b) reduces the mechanical efficiency of the respiratory muscles by affecting their length-tension relationship and c) by combining the two previous points, potentially induce respiratory muscle fatigue and dyspnea.

Entrainment

Entrainment or synchronisation between limb movements and breathing pattern is commonly observed in exercising humans and quadrupeds (Asmussen, 1964; Bramble & Carrier, 1983). In humans, entrainment has been reported during cycling, running and rowing (Bechbache & Duffin, 1977; Kohl *et al,* 1981; Paterson *et al,* 1986; Szal & Schoene, 1989) and, although the exact "stimulus" for entrainment remains obscure, several factors are associated with its occurrence. Contraction of the thoracic musculature is required both for respiration and for stabilisation of the trunk during locomotion. Consequently, it is important that breathing and locomotion are synchronised so that the one does not interfere with the other (Bramble & Carrier, 1983). The physiological benefit from such coupling could be an improvement in the efficiency of the respiratory muscles, possibly improving gas exchange and deterring the development of diaphragmatic fatigue (Johnson *et al,* 1993), or reducing respiratory sensation. Indeed, locomotion may influence the efficiency of respiratory activity or conversely, respiratory muscles may modulate locomotory functions.

There are three locomotory-associated biomechanical forces, that have been identified in horses, 1) visceral piston movement, 2) foot impact, 3) lumbosacral

flexion and extension. However, their contributions on human respiratory patterns have not been quantified (Bramble, 1989). The impact of abdominal compressive forces, secondary to lumbosacral flexion and extension, on breathing patterns has been examined in studies of elite rowers. The cramped body position assumed by the rower at the start of the rowing stroke has been thought to impair diaphragmatic excursions (Cunningham *et al,* 1975), and that was expected to affect the pattern of breathing adopted by the rowers. However, Siegmund *et al* (1999) suggested that a greater limitation of diaphragmatic function exists in the finish position than in the catch. This limitation was attributed to the transient abdominal pressure induced by co-contraction of the diaphragm and abdominal muscles at the finish to stabilise the upper body. This was supported by Manning *et al* (1998) who reported that expiring during the drive phase creates a higher intra-abdominal pressure at the mid-drive position. They suggest that the high levels of shear and compression to which the lower lumbar vertebrae are exposed may be partially compensated by the high intra-abdominal pressure. A structural manifestation of what is essentially a Valsalva manoeuvre is the cardiac hypertrophy found in rowers (Secher, 1993; Clifford *et al*, 1994).

Steinacker *et al* (1993) studied oarsmen performing incremental rowing ergometry. They found that as work intensity increased, rowers shifted from a 1:1 to a 2:1 breath/stroke ratio. They concluded that this transition was due to pulmonary mechanical constraints. As the demand for tidal volume increased with increasing work intensity, breathing efforts encroached upon the flat portion of the pulmonary compliance curve. Hence, a greater portion of the negative intrapleural pressure change is used to overcome pulmonary elastic work. However, although the transition from a 1:1 to a 2:1 ratio circumvented the mechanical limitation of lung distension during inspiration, the respiratory system was now confronted with a different mechanical constraint, that of expiratory flow limitation.

Interestingly, given that such mechanical limitations developed, one would have predicted that the onset of inspiration relative to the stroke cycle would have been tightly regulated. This was specifically examined by Mahler *et al* (1991a), who studied the breathing pattern of elite oarswomen. They found that, whereas the rowers consistently entrained breathing and stroke frequencies 1:1 or 2:1, considerable variability in the onset of inspiration during the rowing cycle occurred. Some rowers initiated inspiration prior to the "catch" or start of the cycle, some inspired at the point of the catch, and others inspired at the finish of the stroke cycle. Thus, despite the development of a significant biomechanical limitation, optimisation of the onset of either inspiration or expiration was not apparent. A possible explanation for this finding may be the incremental nature of the rowing protocol used and the variable level of rowing skill and experience of the subjects used.

In a resent study (Siegmund *et al*, 1999) investigating entrainment during a simulated 2,000m race, rowers were found to breathe at similar times in the stroke cycle. Inspiration occurred most frequently during the first 40% of recovery, followed by expiration during the latter part of recovery. It was also suggested that there are advantageous times in the stroke for large inspired and expired volumes and rowers appeared to be taking advantage of them. Breaths are taken at times where muscle synergy produces larger volumes for a given amount of respiratory work, or alternatively, the same volume for less respiratory work.

1-1.3 Conclusions

The available data support the behavioristic approach to breathing in exercise advocated by Wallace Fenn (1963). Even though it has long been recognised that the breathing pattern is being optimised, the variables being minimised are uncertain. Fenn's main point was that the breathing pattern is modified in order to achieve the greatest comfort. It seems that during competitive rowing the support of external work is far more important than achieving greater comfort. Even though

previous reports speculated that breathing drives locomotion during rowing, recent findings support the theory that locomotion drives ventilation (Siegmund *et al*, 1999).

From the foregoing it is also evident that there is a discrepancy in the literature as to whether rowers hypoventilate, due to the mechanical constraints secondary to the entrained breathing pattern, or develop an appropriate hyperventilatory response during simulated rowing. In a recent study, Dempsey *et al* (1998) suggested that $\dot{V}_E$ of female subjects is increased when breathing a Helium mixture (HeO_2) during high intensity exercise due to a reduction in expiratory flow limitation. These data provide evidence that even relatively small amounts of expiratory flow limitation have an inhibitory effect-acting via reflex feedback inhibition from narrowed airways- on the magnitude of the ventilatory response during exercise in women. Even though rowing was not the exercise modality used in the above study, it suggests that a similar inhibition of the ventilatory response exists during rowing when expiratory flow limitation occurs. The forgoing data also highlight the importance of breathing in rowing. The following section will examine evidence for an influence of ventilatory factors upon exercise tolerance

1-2 Ventilatory Factors Limiting Exercise Tolerance

The following section presents a brief presentation of the pathways through which the function, or dysfunction, of the respiratory pump can directly, or indirectly, affect exercise tolerance. Factors that may limit exercise tolerance include the cardiovascular consequences of exercise hyperpnea, the exercise-induced arterial hypoxaemia, the exercise-induced respiratory muscle fatigue and the dyspneic sensations of heavy exercise

1-2.1 Cardiovascular Consequences of Exercise Hyperpnea

Oxygen Cost Associated With Exercise Hyperpnea

The cardiovascular and pulmonary systems, which support the muscle-tissue requirements for gas exchange and acid-base regulation, themselves require increased blood flow and gas exchange. At high work rates, a progressively greater component of total body energy exchange is required to "support" these systems.

The increased elastic and resistive work of rowing hyperpnea, described previously, not only affects the breathing pattern but also increases respiratory muscle $\dot{V}_{O_2}$ (McCool *et al,* 1986). Additionally, it is likely that extra work is performed by the respiratory muscles during deformation of the rib cage and abdominal walls (Goldman *et al,* 1976). Indeed, a study investigating the factors involved in rib fracture incidents in rowers found that there was a sudden compression of the ribcage displayed after the catch during the first part of the leg drive (which was greater when the rower was breathing), which was not simultaneous with chest wall muscle activity (Wasjwelner, 1996).

A series of studies investigating the oxygen cost of breathing during incremental exercise have shown an exponential increase in the metabolic requirements of the respiratory muscles with progressive hyperventilation. Indeed, these requirements comprised a greater and greater share of the rise in the total body $\dot{V}_{O_2}$ (Aaron *et al,* 1992a; 1992b). As a result, the respiratory oxygen cost at $\dot{V}_{O_2max}$ intensity was about 8%-10% of the total $\dot{V}_{O_2}$, but with a very large variability, with values also in the range of 13%-16% of $\dot{V}_{O_2max}$. Interestingly, the subjects demonstrating these higher values had greater than average $\dot{V}_{O_2max}$, experienced significant expiratory flow limitation, had very high levels of inspiratory and expiratory work of breathing,

and had to perform substantial additional work on a distorted chest wall. The above description fits the profile of the elite rower

Effects of Respiratory Muscle Work on Limb Locomotory Blood Flow

The functional significance of such high respiratory metabolic cost was demonstrated in a study by Harms *et al,* (1997) who found a significant effect of the work of breathing during maximal exercise upon locomotor muscle perfusion and $\dot{V}_{O_2}$ in healthy trained humans. Their data suggest that the level of respiratory muscle work normally experienced during maximal exercise in humans attenuates the rise in blood flow and O_2 transport to working locomotor muscles. Experimental manipulations (increase and reduction) of the work of breathing during maximal exercise caused significant changes in locomotor muscle vascular resistance and perfusion. Specifically, the respiratory muscles under load competed effectively with limb locomotor muscles for a significant portion of available total cardiac output at maximal exercise and the size of this redistribution averaged 13% or, ~2 l/min. When the respiratory muscles were unloaded, maximal pulmonary $\dot{V}_{O_2}$ and cardiac output were reduced by 7-10%, whilst leg blood flow and leg $\dot{V}_{O_2}$ increased by 3-5%. These findings suggest that respiratory muscles significantly compete with limb muscles for total cardiac output during maximal exercise.

Implications of Respiratory Work for Exercise Performance

From the above data it can be deduced that when the respiratory muscles are unloaded the legs should be able to do more work. Indeed, a further study by Harms *et al,* (1998) showed that increased power by the legs at $\dot{V}_{O_2max}$ is possible with respiratory muscle unloading. However, in terms of the practical implications of respiratory muscle unloading, very little can be done to influence any of the factors that can minimise the work of breathing (i.e., further minimising airway resistance) since airways are maximally dilated during exercise. However, specific

respiratory muscle training may increase the mechanical efficiency of ventilation, thereby reducing the metabolic requirements of the respiratory muscles (Harms & Dempsey, 1999).

1-2. 2 Exercise-Induced Arterial Hypoxaemia (EIAH)

Definition and Mechanisms

The level of oxygenation in arterial blood during exercise is defined by measurements of arterial P_{O_2} (Pa_{O_2}), HbO_2 saturation, and O_2 content (Dempsey & Wagner, 1999).

EIAH is broadly defined as reduced arterial oxygenation, which may result from a fall in Pa_{O_2} (and thus also in Sa_{O_2}), from a rightward shift of the O_2 dissociation curve without a fall in Pa_{O_2} or from a combination of these processes (Dempsey & Wagner, 1999).

Specifically, Pa_{O_2} is determined by the level of alveolar ventilation at any given metabolic demand, together with the efficiency with which O_2 is exchanged between alveolar gas and arterial blood, as indicated by the alveolar-to-arterial P_{O_2} difference ($A-aD_{O_2}$). Arterial O_2 saturation (Sa_{O_2}) follows Pa_{O_2} but may be modified by O_2 dissociation curve shifts caused by changes in pH, Pa_{CO_2}, and blood temperature during maximal exercise. Arterial O_2 content (Ca_{O_2}) follows saturation but may be modified by the slight increase in Hb concentration developing from rest to heavy exercise.

Reductions in Pa_{O_2} during maximal exercise in highly fit male subjects have been linked to an excessive alveolar-to-arterial oxygen difference which can reach 20 to 30 mm Hg or, in severe cases, 35 to 40 mm Hg (Dempsey *et al*, 1984). Three potential mechanisms have been thought to be responsible for the EIAH: 1)

insufficient compensatory hyperventilatory response, 2) ventilation-perfusion inequalities and 3) a diffusion impairment.

Variations in O_2 saturation at maximum exercise have been predicted from a multiple linear regression model, where ventilation (as reflected by Pa_{CO_2}) explains 60% of the variance in Sa_{O_2}, $\dot{V}_{O_2max}$ accounts for 25% of it, and $A-aD_{O_2}$ for the remainder (Dempsey & Wagner, 1999). It is beyond the scope of this thesis to further elaborate on all the factors involved in the mechanisms responsible for the EIAH which is a multi-factorial phenomenon. For the purpose of providing background information it will suffice to acknowledge that even though EIAH cannot be completely prevented by increasing alveolar ventilation an adequate hyperventilatory response should minimise the $A-aD_{O_2}$ and mitigate falls in hemoglobin saturation.

Evidence of EIAH

Exercise-induced arterial hypoxemia has been well documented in elite young male athletes (Dempsey *et al,* 1984; Powers *et al,* 1992), older athletes (Prefaut *et al,* 1994) and healthy young women (Harms *et al,* 1998). The EIAH found in many active healthy young women was at a $\dot{V}_{O_2max}$ substantially less compared with their male counterparts. The authors suggest that the smaller lung volumes and diffusion surface, narrowed airways, and even reduced levels of circulating haemoglobin in healthy women provide a morphological basis for gender differences in both diffusion capacity and in the uniformity of intra-regional distribution of ventilation (Harms *et al,* 1998). Futhermore, an argument is made for the presence of mechanical limitation in the hyperpneic response to maximal exercise. Additional data from the same group suggests that the existence of a mechanical limitation in the ventilatory response is further supported by the presence of expiratory flow limitation in female athletes (McClaran *et al,* 1998). The commencement of EIAH during submaximal exercise lead these authors to

favour the $\dot{V}_A / \dot{Q}_C$ maldistribution as the responsible mechanism for EIAH in women (Harms *et al,* 1998).

Support for a hypoventilatory mechanism comes from an earlier study by the same group (Johnson *et al,* 1992). A borderline effective (Pa_{CO_2} 35-38 mmHg) or an absent hyperventilatory response (Pa_{CO_2} >38 mmHg) may indirectly exacerbate the arterial hypoxaemia in the sense that alveolar P_{O_2} is prevented from increasing to a very high value to compensate for the excessively widened alveolar-to-arterial difference and prevent Pa_{O_2} from falling (Johnson *et al,* 1992). Indeed, the recent report of Durand *et al* (2000) provides additional support for this mechanism. All the highly trained male athletes in this study demonstrated a decrease in Pa_{O_2} and a relative hypoventilation during the submaximal stages of an incremental test. However, not all athletes developed EIAH during the maximal stages of test. The authors concluded that the degree and the time of onset of the hyperventilatory response was the determinant factor for the development of EIAH in their subjects.

The reasons for the relative hypoventilation, present even at sybmaximal exercise, are not clear. It has been proposed that the ventilatory control system in hypoxaemic athletes is less 'responsive' (or sensitive) to a given sensory stimulus throughout all exercise intensities (Harms *et al,* 1998). Whether or not these stimuli are the chemoreceptor feedback influences, the locomotor-linked stimuli responsible for 'exercise hyperpnoea', per se, or their combination is unknown. Hypoventilation has also been related to the training volume of the athletes (Durand *et al,* 2000). Indeed, these authors suggest that athletic training may reduce the chemoresponsiveness of the ventilatory control system and result in a relative hypoventilation for a given metabolic rate.

Another explanation for the relatively small hyperventilation of highly trained subjects may be the mechanical constraints on $\dot{V}_E$ during maximal exercise.

Significant feedback inhibition of respiratory motor output during heavy exercise has been suggested following dynamic hyperinflation and increase of end-expiratory lung volume (EELV). A rise in EELV could inhibit the inspiratory motor output via lung stretch at high end-inspiratory lung volume (McClaran *et al,* 1999).

Exhaustive rowing has been associated with marked hypoxaemia. In cases where a marked hyperventilation is present, arterial oxygen tension declines from a PO_2 of 105mmHg at rest to 88 mmHg during the last minute of maximal simulated rowing (Clifford *et al,* 1990). However, Rasmussen *et al* (1991), who found a haemoglobin saturation of 91% during simulated rowing, reported a slightly elevated arterial oxygen tension subsequent to the presence of a moderate hyperventilation. These authors suggested that pulmonary factors were not responsible for the observed arterial desaturation. A more recent report by Nielsen *et al* (1998) found saturation levels below 90% following maximal rowing. Even though in some subjects Pa_{CO_2} remained elevated (39 mmHg), the authors concluded that hypoventilation was not evident since the minute ventilation and alveolar oxygen tension were not compromised.

Consequences of EIAH for Performance

EIAH reduces $\dot{V}_{O_2max}$. The threshold of desaturation at which this effect is measurable is somewhat variable among subjects, but a consistent effect appears to be initiated at 3-4% O_2 desaturation below resting levels (Wagner *et al,* 1991). The further reduction of $\dot{V}_{O_2max}$ beyond this threshold of desaturation changes linearly with Sa_{O_2} (and Ca_{O_2}) such that $\dot{V}_{O_2max}$ is affected by 15% in subjects who desaturate to a maximum of 85–90% Sa_{O_2} at $\dot{V}_{O_2max}$. This effect of EIAH on $\dot{V}_{O_2max}$ is theoretically based on the reduction in Sa_{O_2} and Ca_{O_2} and consequently, on the limits placed on the widening of the maximal arterial-to-venous O_2 content difference across the working muscle.

A study by Nielsen *et al* (1998) investigated the effect of desaturation upon rowing performance. Following the administration of a hyperoxic mixture the severe desaturation to 85% was alleviated and $\dot{V}_{O_2max}$ increased by 13%. However, the effect on rowing performance, which increased by a non-significant 3%, was minimal. (Nielsen *et al,* 1998). The authors suggested that the elevated $\dot{V}_{O_2}$ may reflect enhanced metabolic rate in 'non-exercising' tissues. These data also suggest that $\dot{V}_{O_2max}$ is not a sensitive index for monitoring improvements in rowing performance.

In conclusion, there is no supporting evidence that minimising EIAH through an enhanced hyperventilatory response would be beneficial to rowing performance. If anything, the additional ventilation required to improve the saturation of hemoglobin is likely to be at a very high metabolic cost, in terms of respiratory muscle blood flow, that could adversely affect performance. It seems that hypoventilation may be a beneficial strategy in elite rowers. Indeed, a reduction of the metabolic cost of breathing may spare a fraction of the total cardiac output, otherwise allocated to the respiratory muscles, and improve peripheral muscle perfusion and consequently performance.

1-2.3 Exercise-Induced Respiratory Muscle Fatigue

Although entrainment and the associated changes in breathing pattern are of interest in their own merit, they also exert important influences on the "operational strength" of the inspiratory muscles (Clanton *et al,* 1985). "Operational strength" is defined as the capacity to achieve pleural pressure (P_{pl}) under the conditions that exist for the given tidal volume (V_T) and inspiratory flow (V_I). The pressure-flow relationship in the intact respiratory system is a reflection of the force-velocity relationship of the individual skeletal muscles (Agostoni & Fenn, 1960). According to Hill (1938), for the same level of activation, a muscle's ability to generate tension decreases as its velocity of shortening increases. Leblanc *et al* (1988) confirmed the original descriptions of Agostoni & Rahn (1960) according to which strength declines by 1.7% for every 1% of vital capacity increase in V_T and by 5% for each 1L/min increase in flow. Thus, the pressure or force developed by the inspiratory muscles, may approach 90% of their capacity at peak exercise in fit subjects.

P_{pl} may be expressed as a fraction of the subject's maximum capacity ($P_{pl\,cap}$) to generate that pressure ($P_{pl}/P_{pl\,cap}$). $P_{pl\,cap}$ varies with muscle length and velocity of shortening, which change with lung volume and respiratory flow, respectively. Clearly, inspiratory P_{pl} (P_{pl} I) varies throughout the breathing cycle and a more representative index of respiratory muscle activity is given by the mean $P_{pl}/P_{pl\,cap}$, or tension-time index (TTI), which is the $P_{pl}/P_{pl\,cap}$ averaged over the respiratory cycle (Bellemare & Grassino, 1982). The TTI is defined as the product of the ratio of mean pressure to maximal pressure generating capacity and the inspiratory duty cycle (T_I /T_{TOT}).

In a similar fashion to P_{pl}, diaphragmatic pressure (P_{di}) has been expressed as a fraction of $P_{di\,cap}$. The tension-time index of the diaphragm (TT_{di}) is a major determinant of the energy cost of breathing and of susceptibility to fatigue. It has been suggested that once TT_{di} (T_I /T_{TOT} * $P_{di}/P_{di\,max}$) exceeds 0.15-0.18 the

development of muscle failure is related to the duration of contractile activity (Bellemare & Grassino, 1982). The inherent difficulties with TT_{di} during exercise is that the P_{di} is a dynamic measurement expressed relative to a static measurement of $P_{di\,max}$. Furthermore, $P_{di\,max}$depends on the conditions of both the chest and the abdominal cavities at the time of measurement. If the abdomen is relaxed, $P_{di\,max}$ is equal to the maximal mouth or esophageal inspiratory pressure. However, increased pressure in the abdomen increases $P_{di\,max}$ due to reduced compliance of the abdominal wall.

It is also inherent in the definition of TT_I that fatigue is an ongoing process which is initiated once a recruitment threshold is passed. At this state a reduction of force is observed without the presence of the ultimate "task failure". Therefore, the original definition of Edwards (1981) of skeletal muscle fatigue as a "failure to maintain the required or expected force" has been extended for respiratory fatigue to include also the state of muscle weakness during which an additional respiratory motor output is required for a given force. (National Heart, Lung and Blood Institute, 1990).

Assessment of Fatigue

The identification of fatigue in respiratory muscles is not as clear as with skeletal muscle, making it difficult to identify whether respiratory muscle fatigue limits exercise, or influences the pattern of breathing. The development of a rapid shallow breathing pattern has been suggested as an indirect indicator of respiratory muscle fatigue (Gallagher *et al,* 1985). However, the adoption of this breathing pattern during exercise may also signify an attempt to minimise effort in the face of developing fatigue, rather than a consequence of fatigue itself.

The maximum voluntary mouth pressure measurement, used to indirectly monitor the progressive decrease in the force output capacity at a given muscle length, is the most commonly used technique (Black & Hyatt, 1969). Reductions of

maximum mouth pressure interpreted as evidence for respiratory muscle fatigue following exercise have been found (Mahler & Loke, 1981; Loke & Virgulto, 1982; Bye *et al*, 1984). A number of methodological considerations such as the variability in response to repeated measurements, the day to day biological variability similar to any measure of strength, and the learning effect, have rendered this technique vulnerable to criticism and the conclusions of these studies open to debate. However, these measurements, when performed by motivated individuals, have provided very useful information about the overall strength of the respiratory muscles, which is relevant to the ability of the system to meet the ventilatory demands (Black & Hyatt, 1969).

Bilateral phrenic nerve stimulation (BPNS) and concomitant measurements of trans-diaphragmatic or airway occluded mouth pressures can also be used to assess diaphragmatic fatigue. Although several other respiratory muscles are recruited with whole-body exercise (i.e., external intercoactals, scalenes and sternocleidomastoid muscles), the diaphragm is the primary inspiratory muscle and the most effective pressure generator; thus providing the best index of respiratory system muscular function. Initially, the practical difficulties associated with this technique raised some doubts as to its applicability during exercise (Levine & Hanson, 1988). The results of Levine and Hanson's study suggested that the human diaphragm was not fatigable with short-term progressive exercise to exhaustion, unless a resistive load was added. However, in a later study, significant reduction in the twitch trans-diaphragmatic pressure (P_{di}) ranging between 15% and 26% was found following exercise to exhaustion at a power output equivalent to 80%-95% of $\dot{V}_{O_2max}$ (Johnson *et al,* 1993). Thus, BPNS assessment of diaphragmatic function supports the findings of studies using voluntary measurement of global inspiratory muscle strength (Mahler & Loke, 1981; Loke & Virgulto, 1982), (i.e.), inspiratory muscles are susceptible to fatigue.

Factors Influencing Exercise-Induced Respiratory Fatigue

It seems from the above that the development of fatigue may be related to the combination of intensity and duration of the performed exercise. There has also been a suggestion that the fitness level of the subjects may play a role in the occurrence of diaphragmatic fatigue during exercise. Specifically, it has been suggested that the respiratory muscles of 'athletic' individuals have superior strength and greater fatigue resistance (Coast *et al,* 1990). However, BPNS provided a contrasting view since no difference was found in the degree of diaphragmatic fatigue between highly and average fit subjects post exercise at 95% of $\dot{V}_{O_2max}$ (Johnson *et al,* 1993).

McConnell *et al* (1997) showed that even though respiratory muscle fatigue is independent of whole-body fitness level, a relationship exists between baseline maximum inspiratory mouth pressure (MIP) and percentage fall in MIP post exhaustive exercise. They concluded that individuals with the stronger inspiratory muscles showed significantly smaller reductions in MIP than the individuals with modest baseline capacity. In this regard a comparative study between rowers and normal subjects found greater respiratory muscle pressures in the rowers (Donnely *et al*, 1991). Donnely *et al* concluded that their data argue against the development of respiratory muscle fatigue in rowers.

Significance of Respiratory Muscle Fatigue to Exercise Performance

The development of respiratory muscle fatigue could limit exercise in different ways. First, a greater respiratory motor command must be produced for the higher activation of the respiratory muscles required to maintain the same ventilatory output. An increased respiratory motor outflow, being the major determinant of the sense of respiratory effort, will considerably increase dyspnea (Altose, 1986; Killian and Campbell, 1986). Consequently, exercise could be terminated by dyspneic sensations, or because of the contribution dyspnea makes to perceived exertion. If activation of the respiratory muscles is not increased, then

hypoventilation will occur and exercise may be terminated by unfavorable gas exchange and acid-base balance. Finally, the adoption of a rapid shallow pattern of breathing could further affect gas exchange and interfere with the entrainment of the breathing with the locomotory pattern. Such an interference could affect exercise by decreasing the mechanical efficiency of locomotion.

However, fatigue of the diaphragm can occur without compromising the ventilatory response to exercise (Babcock *et al,* 1995). A study in a group of rowers examined the effect on exercise performance of unloading the respiratory system by helium mixture breathing (Aaron *et al,* 1985). It was found that at an intensity of 90-95% $\dot{V}_{O_2max}$ the time to exhaustion increased by 40% while breathing helium. Later, studies using mechanical pressure assist for unloading of the respiratory muscles have also shown increases in exercise tolerance (Harms *et al,* 1998). Even though this design did not ascertained whether the increase in exercise tolerance was due to avoiding respiratory fatigue per se, it clearly showed that the respiratory load is important to the exercise tolerance, or dyspnea, or breathing pattern.

Studies that have compared exercise performance with and without prior respiratory fatigue suggest that reduced ventilatory capacity alone is sufficient to decrease short term maximal exercise performance. Martin *et al,* (1982) found that running performance was reduced from 7.6 min in the 'fresh' state to 6.5 min following the induction of respiratory fatigue with 150 min of isocapnic hyperpnea. This study may be particularly relevant to rowing since the performance times are comparable to competitive rowing.

More recent studies by Mador & Acevedo (1991a, 1991b) showed similar results following respiratory fatigue induced by resistive loading to failure with a pressure threshold of 80% of maximal mouth pressure. Cycling times to exhaustion at 90% of maximal capacity were reduced from 311 ± 96 to 238 ± 69 (SD)s. Following the

induction of respiratory fatigue, the cycling trials were characterised by a rapid breathing pattern and increased respiratory sensation.

In conclusion, during rowing the combination of the entrained breathing pattern, the mechanical limitations of the pulmonary system and the additional static supportive work for the upper body, place high demands upon the respiratory muscles. These demands predispose the respiratory muscles to fatigue despite of the high fitness levels observed in rowers. Due to the various implications that respiratory muscle fatigue can have upon rowing performance, the aim of part of this thesis will be: a) to rigorously investigate the incidence of respiratory muscle fatigue during rowing, b) to reduce any respiratory muscle fatigue by means of inspiratory muscle training, and c) to evaluate the effect of such an intervention upon rowing performance.

1-2.4 Dyspnea

Definition

Dyspnea has been defined as: "difficult, labored, uncomfortable breathing" (Wright & Branscomb, 1954), "awareness of respiratory distress" (Wasserman & Cassaburi, 1988), "the sensation of feeling breathless or experiencing air hunger" (Simon *et al,* 1989), or as "an uncomfortable sensation of breathing" (Mahler *et al,* 1996). However, these definitions sometimes mix the true symptom (i.e., what the subjects say) with physical signs (i.e., what the investigator observes about the subject, e.g., "exhibits labored breathing"). Nevertheless, a symptom can only be described by the person who experiences it and in this context, recent investigations of the perception of breathlessness suggest that there are several types of dyspnea (Simon *et al,* 1990; Schwartzstein & Cristiano, 1996; Mahler *et al,* 1996). The sensation of air hunger or breathlessness has been associated with

stimulation of the chemoreceptors (Banzett *et al,* 1989) while the sense of effort to breathe or dyspnea is believed to reflect central respiratory motor command (El-Manshawi *et al,* 1986). Even though there is an argument that dyspnea associated with pathological states should be regarded as distinct from the breathlessness reported by healthy subjects on exertion, there is no good evidence that the sensory experiences are different.

In the 1950s and 1960s much of the work on dyspnea focused on the impact of mechanical loads on respiratory symptoms (Howell & Campbell, 1966). While there was an awareness that there may be several different qualities of dyspnea, the general consensus was that the sense of effort was the primary factor of breathing discomfort. In the past decade, great steps have been made to distinguish the sensations included in the term dyspnea and in defining a vocabulary to facilitate communication between subjects and investigators about these sensations (Simon *et al,* 1990; Schwartzstein & Cristiano, 1996; Mahler *et al,* 1996). We now have a greater appreciation for the differences between a respiratory "sensation," the neural activation resulting from stimulation of a peripheral receptor, and "perception," the reaction of the sentient individual to the sensation (Guz, 1997).

Psychological and cultural factors may influence the reaction to a sensation, e.g., a stoic individual may deny respiratory discomfort and push beyond the limitations experienced by another person more sensitive to bodily messages. Indeed, taking part in competition is always associated with psychological stress and, therefore, psychophysical resilience is one of the most important aspects of the long preparation of athletes for high level performance. The context in which a sensation occurs can also influence the perception of the event. The sensation experienced by an individual during maximal exercise and/or competitive situations may evoke very different reactions than the same sensation occurring at rest.

It has been suggested that dyspnea is a term used to characterize a subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity. This experience is derived from interactions among multiple physiological, psychological, social, and environmental factors, and may induce secondary physiological and behavioral responses. This broad definition of dyspnea will be used herein.

Mechanisms of Dyspnea

Physiological

The sensation of dyspnea seems to originate with the activation of sensory systems involved with respiration. Sensory information is, in turn, relayed to higher brain centers in the sensory cortex where central processing of respiratory related signals and contextual, cognitive, and behavioral influences shape the ultimate expression of the evoked sensation. Respiratory motor activity emanates from clusters of neurons within the brainstem in the medulla. Feedback modifying this activity is projected by chemoreceptors in the vasculature/carotid and brain, mechanoreceptors in the airways, lungs, and chest wall. Furthermore, these receptors project a sensory input and may have a direct dyspnogenic effect.

Additionally, corollary signals or efferent copies of brainstem respiratory center motor output are transmitted to higher brain centers and result in a conscious awareness of the outgoing motor command (McClosky, 1978). These corollary discharges are thought to be important in shaping the sense of respiratory effort. It is well established that factors that necessitate a greater motor command to achieve a given tension in the muscle, such as decreasing muscle length, muscle fatigue, or respiratory muscle weakness, cause a heightened sense of respiratory effort (Killian *et al*, 1984; Campbell *et al,* 1990; Supinski *et al,* 1987). The sense of

respiratory effort intensifies with increases in central respiratory motor command and is proportional to the ratio of the pressures generated by the respiratory muscles to the maximum pressure–generating capacity of those muscles (El-Manshawi *et al,* 1986).

Conditions affecting dyspnea

1. Elevated Ventilatory Demand

Dyspnea intensifies in parallel with exercise minute ventilation both in normal individuals and in patients with lung disease (Killian *et al,* 1992). This has been attributed to the increase in respiratory motor output and to a corresponding increase in the sense of effort. While the level of ventilation often correlates well with the intensity of dyspnea, increases in central inspiratory activity alone are unlikely to explain respiratory discomfort in all settings (Manning , 1998). An example of this mismatch is the paradox observed immediately following the termination of exercise when ventilation is dropping sharply and dyspnea increases. Indeed, a key observation for the understanding of neurophysiological basis of dyspnea is that isocapnic voluntary hyperventilation to a level associated with substantial discomfort during exercise or hypercapnia causes little or no discomfort (Adams *et al,* 1985). Thus, it appears that dyspnea depends on the nature of the ventilatory stimulation (i.e., automatic vs. voluntary).

2. The theory of "length-tension inappropriateness"

When changes in respiratory pressure, airflow, or movement of the lungs and chest wall are not appropriate for the outgoing motor command, the intensity of dyspnea is heightened. In other words, a dissociation between the motor command and the mechanical response of the respiratory system may produce a sensation of respiratory discomfort. This theory, which was first introduced by Campbell and Howell in the 1960s, provides a unifying mechanism by which the dyspnea in many pathological conditions such as respiratory muscle abnormalities, altered chest wall mechanics and blood-gas abnormalities is

explained. Respiratory diseases that usually cause dyspnea include asthma and COPD, which narrow airways and increase air-way resistance, as well as, diseases of the lung parenchyma, such as interstitial pneumonitis and pulmonary fibrosis, which increase lung elastance.

Assessment of Dyspnea

Dyspnea, like hunger or thirst, is largely a "synthetic sensation" in that it often arises from multiple sources of information rather than from stimulation of a single neural receptor. In addition, the severity of dyspnea as well as the qualitative aspects of unpleasant breathing experiences varies widely among subjects. The variable nature of dyspnea reduces the likelihood that any single estimate of organic disease, or illness, or exercise performance will provide a fixed index either to establish the intensity of dyspnea or to evaluate the success of a given therapy. Therefore, dyspnea itself needs to be measured. Any assessment of dyspnea must consider whether we are trying to measure the intensity, quality of the sensation or the emotional or behavioral response to the discomfort.

Measurements of dyspnea during exercise can be examined in relation to workload, power production, maximal oxygen uptake, or interactions among a range of respiratory-related variables. In normal subjects, dyspnea intensifies as the oxygen uptake and carbon dioxide output increase with muscular activity. The intensity of dyspnea is considered appropriate when the ventilation is increased or when the ventilatory capacity is reduced. Respiratory muscle effort intensifies with ventilation as the load opposing inspiratory muscle contraction increases or when the inspiratory muscles are intrinsically weak or weakened by hyperinflation or fatigue.

The exertional activity of the inspiratory muscles depends on the neural activity responding to metabolic demand; the mechanical properties of the muscles,

including length and strength of the inspiratory muscles; the endurance of the muscles, which depends on the ability of the heart and blood to supply oxygen to the tissue in combination with the nutritional and metabolic capacities of the muscles, and the load against which the muscles must contract.

Maximal ventilation requires repetitive maximal inspiratory muscle contraction, and this results in fatigue. More effort in terms of motor unit recruitment and/or activation is required to achieve the same ventilatory task if the activity is sustained. Therefore, fatigue may contribute to the intensity of dyspnea experienced during sustained effort. The formal measurement of dyspnea during incremental exercise to symptom-limited capacity has become increasingly popular in the investigation of dyspnea. Ventilatory capacity is measured prior to exercise, ventilation is measured during exercise, and these are related to the intensity of dyspnea. Even though other scales exist the most popular in exercise physiology studies are the Borg Scale and the Visual Analogue Scale.

The Borg Scale

Borg (1970) first described a scale ranging from 6 to 20 to measure perceived exertion during physical exercise. The scale was subsequently modified to a 10-point scale with verbal expressions of severity anchored to specific numbers (Altose and Cherniack, 1981; Borg, 1976) (fig. 1.1). Additional terms at the ends of the scale anchor the responses, thus facilitating more absolute responses to stimuli and enabling direct inter-individual comparisons (Borg, 1973; Borg, 1982). The scale is open at the high end to accommodate for the ceiling effect and improve the reliability. Care must be taken to provide consistent, specific instructions when using the scale. For example, different investigators have asked subjects to rate "severity of breathlessness," "need to breathe," and "effort of breathing". Extensive reports demonstrate the reliability and validity for Borg ratings of dyspnea from 1 day to 1 year. (Adams *et al,* 1985a; El-Manshawi *et al,* 1986; LeBlanc *et al,* 1986; Silverman *et al,* 1988; Skinner *et al,* 1973). Normative

data are available for the Borg scale during incremental cycle ergometry (Killian *et al*, 1992).

HOW WOULD YOU RATE YOUR BREATHLESSNESS ?

0	Nothing at all
0.5	Very, very slight (just noticable)
1	Very slight
2	Slight
3	Moderate
4	Somewhat severe
5	Severe
6	
7	Very severe
8	
9	Very, very severe (almost maximal)
10	Maximal

Fig 1.1 The modified Borg scale for breathlessness (Borg, 1982).

The Visual Analogue Scale (VAS)

The VAS consists of a line, usually 100 mm in length, placed either horizontally or vertically on a page, with anchors to indicate extremes of a sensation (fig 1.2)(Gift, 1989). The subject judges intensity by marking a point on the line (or moving a pointer), and distance along the line is taken as the measure of sensation. It is a continuous interval scale with very high resolution and it has been be used to monitor the time course of changing sensations (Adams *et al,* 1985) with good reliability and validity (LeBlanc *et al,* 1986; Adams *et al,* 1985a). Common problems encountered in administering the VAS are difficulty seeing the line and anchors as well as forgetting how the scale is oriented.

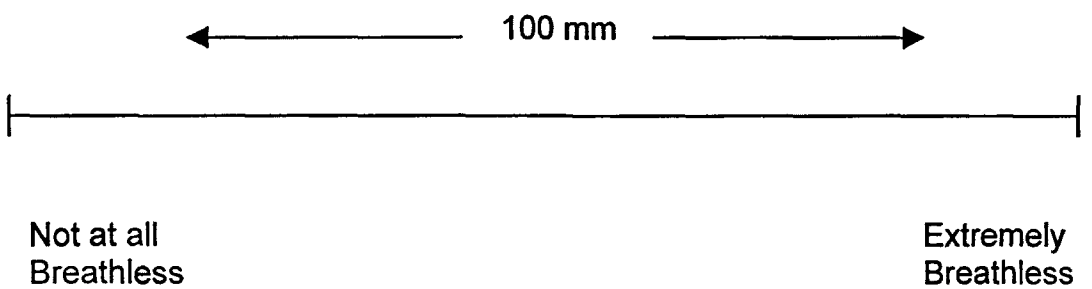

Fig 1.2 The Visual Analogue Scale (VAS)

Treatment of Dyspnea

From the above it can be deduced that any therapeutic intervention that reduces ventilatory demand, reduces mechanical loading, or strengthens inspiratory muscles, should relieve dyspnea by reducing the required motor output and/or by reducing fatigue.

Reduced Ventilatory Requirement

Interventions that reduce CO_2 output ($\dot{V}_{CO_2}$), physiologic dead space (V_D/V_T), arterial hypoxemia, metabolic acidosis or alter the set point for arterial CO_2, will reduce $\dot{V}_E$ and dyspnea at a given work rate during exercise. Dyspnea improvement is multifactorial, but in a study that used regression analysis with multiple relevant independent physiologic variables, reduced $\dot{V}_E$ per work rate slopes emerged as the only significant predictor of change in Borg ratings following exercise training in patients with chronic obstructive pulmonary disease (COPD)(O'Donnell *et al,* 1995). Reduction in $\dot{V}_E$ was achieved primarily by a reduction in breathing frequency with little change in tidal volume (V_T) (O'Donnell *et al,* 1995). In this setting, reduced ventilatory demand is likely related to improved efficiency, seen as $\dot{V}_{CO_2}$ and oxygen consumption ($\dot{V}_{O_2}$) that are reduced at a given work rate after training (O'Donnell *et al,* 1995). Altered central perception of the breathing discomfort, i.e., desensitization to dyspnea may also account in part for the findings observed in the above study.

Reduced Lung Hyperinflation

The functional residual capacity (FRC) is determined dynamically and not statically in patients with obstructive lung disease and in athletes with expiratory flow limitation (Mota *et al,* 1999). In the setting of expiratory flow limitation and increased ventilatory demand (e.g., during exercise or hyperventilation), the interval between successive breaths is insufficient to re-establish the FRC. In the presence of expiratory flow limitation, expiration is prematurely terminated by a reflex response to dynamic airway compression (Pellegrino *et al,* 1993). Consequently, end-expiratory lung volume is increased above the volume normally dictated by the balance between the recoil forces of the chest wall and lung (i.e., passive FRC). This condition is termed dynamic lung hyperinflation, which has

been shown to have serious mechanical and sensory consequences on the respiratory system.

Dynamic hyperinflation results in the operating V_T being positioned at the upper nonlinear extreme of the respiratory system's pressure–volume relationship, where there is a substantial elastic load. Furthermore, the presence of positive pressure in the airways at the end of exhalation, so called auto–positive end-expiratory pressure (auto-PEEP or intrinsic PEEP), imposes an additional threshold inspiratory load on the ventilatory muscles which must be overcome at the initiation of each breath. Dynamic hyperinflation also results in severe mechanical constraints on tidal volume at rest and at low levels of exercise in patients with severe expiratory flow limitation. McClaran *et al.,* (1999) suggested that similar mechanical constraints occur in competitive athletes during high intensity exercise. Indeed, when the expiratory flow limitation in a group of male cyclists was prevented, by expanding the maximum flow-volume envelope through HeO_2 breathing, both minute ventilation and tidal volume increased significantly.

Dyspnea relief and improved exercise tolerance has been documented following lung reduction surgery (Martinez *et al,* 1997). Improvements have been correlated to the reduced hyperinflation and improved diaphragmatic function.

Improving Inspiratory Muscle Function

Dyspnea has been related to weakness and fatigue of respiratory muscles (Macklem, 1995). As previously mentioned, COPD is characterized by hyperinflation, a condition in which the respiratory muscles must function at a mechanical disadvantage and an increased load due to PEEP. Thus, their ability to generate pressure is reduced. As the pressure generated by the inspiratory muscles per breath approaches the maximal pressure that can be achieved, dyspnea worsens. Reduction of ventilatory demand and impedance will ultimately enhance respiratory muscle function, but specific strategies such as nutritional

supplementation, or inspiratory muscle training can help to maintain or improve respiratory muscle function and add to the overall dyspnea diminution.

a) Nutrition

Alterations in respiratory muscle energy balance, either secondary to a reduction in energy supply to the muscle or an increase in respiratory muscle energy demand (depletion of muscle energy stores) (Braun, 1984) can lead to respiratory muscles weakness, fatigue (Roussos and Macklem, 1982), and increasing dyspnea. Decreased body weight has been associated with decreased diaphragm mass (Arora and Rochester, 1982), intercostal muscle fiber size (Thurlbeck, 1978), and sternomastoid thickness (Arora & Rochester 1984) and fatigability (Efthimiou *et al,* 1988). Several investigators have shown improvement in respiratory muscle function in response to nutritional repletion with short-term use of enteral or parenteral nutrition (Goldstein *et al,* 1986) as well as in controlled trials of outpatient and inpatient oral supplementation with a high caloric diet (Whittaker *et al,* 1990, Rogers *et al,* 1992). However, the choice of outcome measures to assess the impact of nutritional supplementation is controversial and the results of these studies are not conclusive.

b) Inspiratory Muscle Training.

Because of the association between respiratory muscle dysfunction and dyspnea, an improvement in respiratory muscle function with inspiratory muscle training (IMT) could lead to a reduction in dyspnea. A meta-analysis of IMT in 17 clinical trials found limited support for its use in terms of pulmonary function, respiratory muscle strength and endurance, exercise capacity, and functional status in patients with COPD (Smith *et al,* 1992). Nevertheless, the secondary sensitivity analysis of this study reported that in five studies in which strength or endurance of the respiratory muscles did improve a moderate treatment effect on functional exercise capacity was found. Further evidence showed that IMT with resistance breathing leads to a decrease in the intensity of dyspnea. Harver *et al,* (1989)

showed a consistent improvement in clinical baseline and transition dyspnea indices with IMT. Weiner *et al,* (2000) also found improvements in dyspnea following a period of 3 months of specific IMT in patients with asthma. Nield (1999) found reductions in dyspnea during a submaximal cycling test following 6 weeks of pressure threshold IMT in COPD patients.

(See section 1-3 for more detailed description of respiratory muscle training.)

c) Altered Central Perception

Desensitization, or exposure to greater than usual sensations of dyspnea, has been shown to increase a patient's ability to cope with a symptom and potentially heighten the perceptual threshold (Carrieri-Kohlman *et al,* 1993). The precise mechanisms behind these changes in dyspnea, independent of changes in $\dot{V}_E$, are unknown. It is possible that a peripheral adaptation manifested as a decline in the firing rate of peripheral receptors may be responsible. The adaptation has been well documented for vagally mediated lung stretch receptors exposed to a sustained stimulus but there is no evidence that the same holds for the phasic stimulation of ventilation. Exercise training has been proposed as the most powerful means of desensitization to dyspnea (Haas *et al,* 1993).

Implications for Performance

Many attempts have been made by physiologists to explain exercise intolerance as the product of various fundamental physiological/biochemical constraints. The physiological parameters that reach maximum values during maximal exercise have been considered as limiting. When considering peripheral limitations of performance, it is important to recognize that motor activation is also a determinant factor. The intensity of effort resulting from motor activation and the tolerance of the individual to this effort are the final limiting steps in intense muscular activity.

Killian *et al,* (1995) state, "it is evident that people stop exercise at the point of limitation because the discomfort of continuing is intolerable". Indeed, the sensation of peripheral muscle effort combined with dyspnea is a neural event with a sensory and perceptual nature which finally limits exercise tolerance. Tolerance for discomfort varies between subjects. The average healthy subject stops exercise at a symptom intensity rated "very severe" (7 on the Borg psychophysical rating scale)(Killian *et al,* 1992). A symmetry between the sensations of dyspnea and leg effort has been identified. As power output increases the intensity of dyspnea and leg effort increases, until at maximal exercise the intensity of dyspnea and leg effort is high, and one or both are limiting exercise tolerance (Killian, 1987).

In conclusion, it has been suggested that the perceptions of dyspnea and peripheral exertion share the same mechanisms and they both form a composite perception of exertion which ultimately limits performance.

1-2.5 Epitome

The studies presented in this thesis aimed to influence the above mentioned venilatory limiting factors by means of a specific respiratory warm-up and a period of respiratory muscle training. Specifically, we hypothesized that a specific respiratory warm-up and a period of respiratory muscle training may reduce the metabolic requirements of the respiratory muscles for a given work, reduce the exercise-induced respiratory muscle fatigue, increase minute ventilation, and reduce the dyspneic sensations of exercice. Consequently, the metabolic cost of the respiratory muscles, exercise arterial hypoxemia and overall perceived exertion may be reduced and rowing performance may be improved.

1-3. Respiratory Muscle Training (RMT)

It is clear from the preceding sections that inspiratory muscle function and dysfunction have a central role in exercise tolerance. Thus, the question arises, can training the muscles of respiration improve performance? The following section presents a brief overview of previous studies on RMT and performance.

Various regimes of specific respiratory muscle training have previously been utilised to fulfil the principles of overload and specificity required for an adaptive response.

1-3.1 Voluntary Isocapnic Hyperpnea

This form of training involves sustained elevated ventilation. Experimental designs have included simple rebreathing through a dead space to avoid hypocapnia or supplementing oxygen and eliminating carbon dioxide to avoid hypoxemia and maintain isocapnia, respectively. The training stimulus has involved one or more 10-15 min sessions daily at 70-90% of the pre-training maximal voluntary ventilation (Pardy & Rochester, 1992).

1-3.2 Flow Resistive Loading

The load in this form of training is provided by inspiring via flow restricting orifices with variable diameters. The main limitation of these devices is that the inspiratory pressure required varies with the rate of air-flow and not just orifice size. Therefore, a controlled breathing pattern is essential for the implementation of a quantifiable training stimulus (Anderson *et al,* 1979; Kim, 1981).

1-3.3 Elastic Loading

Elastic straps around the rib cage can also be used to train the respiratory muscles by restricting the thoracic excursions and increasing the elastic load on

the chest wall. Similarly to the flow dependent loading, the main limitation of this technique is the quantification of the additional work imposed on breathing.

1-3.4 Pressure Threshold Loading

In threshold loading a device with an opening valve is used as a means of creating resistance. A certain percentage of maximum inspiratory pressure capacity is required to overcome the resistance set by the valve and initiate inspiratory flow. Different designs have used either a weighted plunger or a spring for resisting the opening of the valve (Nickerson & Keens, 1982; Larson *et al,* 1988). The expiration is usually performed through a different valve without the imposition of any load.

In summary, at the current time pressure threshold loading represents the most convenient and most reliable method of respiratory muscle training

The most critical consideration of any muscle training device is the loading profile generated by the imposed resistance. A description of the load profile of the threshold loading device used in the studies presented in this thesis follows. Upon initiation of an inspiratory effort, against a threshold load, the negative pressure generated at the mouth rises continuously until such a point where the threshold load is realised. At this point flow is initiated, and will continue until the time when pressure generation falls below the threshold load. Even though, the total resistance presented by the system is not only the positive force acting on the inspiratory valve but also the flow resistance generated as air passes through the valve, we can ignore the flow component for the quantification of training stimulus since variations in flow during the pressure plateau phase typically have a negligible impact upon the pressure profile observed (Caine, 1998).

1-3.5 Specificity of Respiratory Muscle Training

Previous reports suggest that the principles of force-velocity specificity established for peripheral skeletal muscle training also apply to the conditioning of the respiratory muscles. Specifically, O'Kroy & Coast (1993), and Tzelepis *et al,* (1994a; 1994b) found that training protocols characterised by high inspiratory pressure or high inspiratory flow increase maximal inspiratory pressure or peak inspiratory flow, respectively. Furthermore, flow training improved not only flow but also resistive measures suggesting the existence of a cross over effect between training modalities. Finally, the greatest improvements in inspiratory muscle strength were specific to the lung volume at which training occurred.

1-3.6 Clinical Studies of Respiratory Muscle Training and Exercise Tolerance

The classic study of Leith and Bradley (1976) was the first to show that strength and endurance of the respiratory muscles can be increased following a specific respiratory muscle conditioning protocol. Following this work, the effects of both hyperpnea (Keens *et al,* 1977; Belman & Mittman, 1980) and resistive loading regimes (Anderson *et al,* 1979) were clinically examined. In their meta-analysis, Smith *et al,* (1992) showed that, even though the only significant effect of respiratory muscle training was an increase in maximal voluntary ventilation, the maximal inspiratory pressure, respiratory muscle endurance and functional exercise capacity all improved. Secondary analysis also revealed significant improvements in respiratory endurance, and functional exercise capacity, where flow resistive training with a controlled breathing pattern had been implemented. Furthermore, the clinical data suggest that both the type of intervention utilised and the specific regime implemented are crucial in determining the degree of improvement following training of the inspiratory muscles.

More recent studies in patients with asthma (Weiner *et al.*, 2000) and COPD (Lisboa *et al.*, 1997) utilising pressure threshold loading have indicated positive outcomes for indeces including dyspnea and exercise tolerance.

1-3.7 Respiratory Muscle Training and Exercise Performance in Healthy Subjects and athletes

Some early studies have suggested that respiratory muscle training may have a beneficial effect upon exercise tolerance. Even though no increase in maximum exercise tolerance was observed in the early study of Haas and Haas (1980), submaximal work intensities were associated with lower heart rate and oxygen consumption following 16 days of hyperpnea at 85% peakMIP.

Chen and Martin (1983) showed a significant increase of 6% in treadmill work time at near maximal intensities (~90-95% $\dot{V}_{O_2max}$) following 4 weeks of resistive load training of the inspiratory muscles for 15 min twice daily in a group of normal subjects. However, these improvements in performance were not related to any changes in $\dot{V}_{O_2max}$.

The effect of isocapnic hyperpnea for 30 min per day, 4 days per week for a period of 8 weeks upon exercise performance in healthy elderly people was examined by Belman and Gaesser (1988). These authors reported a significant reduction in perception of dyspnea during a steady-state sub-maximal exercise. Consistent with these findings are those of Copestake and McConnell (1995) who reported similar reductions in dyspnea and a 20% increase in inspiratory muscle strength following pressure threshold training in healthy elderly men and women.

Hanel and Secher (1991) investigated the effects of resistive loading upon the Cooper running test in healthy adults. A comprehensive battery of lung function tests were performed prior and following the 27.5 days training period comprising two daily 10-min training sessions at 50% of the maximum inspiratory pressure.

Since there were no differences in minute ventilation, maximum oxygen uptake, and distance covered in the 12-min run, between the control and the training group, the authors concluded that inspiratory muscle training does not change work capacity. However, in another study by Boutellier and Piwko (1992), the cycle time to exhaustion of sedentary subjects at a submaximal intensity (~64% $\dot{V}_{O_2max}$) was increased by 50% following 4 weeks of daily 30 min hyperpnea sessions.

The first study to examine respiratory muscle training upon exercise performance in trained subjects was by Morgan *et al,* (1987). Although peak minute ventilation was improved, no changes in cycling to exhaustion at 95% of $\dot{V}_{O_2max}$ or $\dot{V}_{O_2max}$ were found following 3 weeks of isocapnic hyperpnea. In a later study by Fairbarn *et al,* (1991), where highly trained cyclists engaged in a total of 16 sessions of isocapnic hyperpnea, time to exhaustion at 90% $\dot{V}_{O_2max}$ increased by 25% in the training group compared with a 4% increase in the control group. Due to the small sample size this difference did not reach statistical significance. No improvements were found in $\dot{V}_{O_2max}$ or $\dot{V}_{E max}$ during an incremental ride to exhaustion.

In another study by Boutellier *et al,* (1992) endurance trained individuals increased their cycling time to exhaustion at the anaerobic threshold (~77% $\dot{V}_{O_2max}$) by 38% following 4 weeks of isocapnic hyperpnea. An investigation of the exercise related metabolic changes following 4 weeks of respiratory training reported a 28% increase in time to exhaustion at the individual anaerobic threshold (Spengler *et al,* 1996). Significant reductions of blood lactate concentrations were observed during the post-intervention cycling trials. Reductions in blood lactate concentrations during submaximal cycling were also found following 6 weeks of IMT (Sharpe, 1999). These reductions in blood lactate concentrations are also reflected in the significantly improved cycling performance reported by Caine (1999). In this study 4 weeks of IMT induced a 32.8% improvement in the time to exhaustion at an

exercise intensity corresponding to approximately 80% $\dot{V}_{O_2max}$. The same study also reported reductions in ratings of perceived exertion. Even though perception of dyspnea was not measured it is reasonable to assume that reductions in dyspnea contributed to the reduction in overall perceived exertion. They were no changes in any of the parameters measured in the control group.

The discrepancies amongst previous studies may reflect methodological differences concerning exercise protocols used in the various studies. One criticism of some of the studies demonstrating a positive effect has been the absence of a carefully matched control group and the use of volitional end points in their testing procedures. Further studies are required to elucidate the effect of inspiratory muscle training upon exercise performance, particularly using performance tests that simulate the competitive environment. These issues will be addressed in this thesis.

1-4. Warm-Up

1-4.1 Introduction

From the foregoing it has become evident that IMT may enhance performance. It is also thought that warm-up aids locomotor function. Therefore, two questions arise a) does warm-up aid inspiratory muscle function to the same extent as locomotor function and b) does a specific respiratory warm-up aid whole body performance?

The period of general preparatory activity before the start of an intense physical activity is known as 'warm-up'. This has been associated with a number of physiological and psychological benefits such as injury prevention (van Mechelen, 1992), control of muscle soreness (Rodenburg *et al,* 1994) enhanced mechanical efficiency (deVries & Housh, 1994), and control of frustration and stress (Anshel, 1993; Ainscoe & Hardy, 1987) prior to important performances. For the asthmatic athlete, the use of warm-up exercises has been shown to significantly decrease post-exercise bronchoconstriction (McKenzie *et al,* 1994). However, the actual warm-up methods (routines) when and if used, are frequently based on the trial and error experience of the athlete or coach, rather than on a scientific study and advice.

Warm-up in its literal meaning implies activities that increase body temperature. This increase results in improvement of certain physiological processes that can be seen from the single muscle fibre to the integrated body systems and performance parameters. For example, temperature increases have distinct effects on aerobic endurance, anaerobic endurance, speed, strength, and power as well as flexibility and co-ordination (Guellich & Schmidtbleicher, 1996; Black *et al,* 1984). However, across the wide spectrum of physical activities and sports these performance-related parameters are rarely seen in their pure form. Usually

they blend together to meet the energy requirements and the movement profile of individual activities.

Warm-up activities have been previously classified as passive, and active (Shellock & Prentice, 1985; Astrand & Rodahl, 1987; Karvonen, 1992; deVries & Housh, 1994).

The active warm-up can be further divided into general and specific warm-up depending on its relevance with the subsequent main activity. The main purpose of the general warm-up is to raise the body temperature and facilitate all the temperature related physiological changes, while the specific warm-up aims to improve the neuromuscular co-ordination and enhance the mental readiness for physical performance. The elevation of body temperature resulting from an active warm-up is due to the heat produced from the transformation of chemical energy into mechanical work during muscle contraction. In contrast, the passive warm-up involves the rise of body temperature with external means such as sauna, hot shower or massage.

1-4.2 Physiological Changes Due to Warm-Up

There are a number of physiological changes due to warm-up, which are summarised in Table 1.1. It is clear that most of these changes are temperature-related phenomena that can be seen in the mechanical characteristics of the muscle fibre, the functional properties of the nervous system and the efficiency of the cardio-respiratory system. At the same time, body warming enhances the heat dissipating activity (Torii *et al,* 1996). In general, preliminary exercises have been shown to allow better adjustments in body temperature and increasing water loss during physical work, which, in turn, reduces the chances of exercise hyperthermia and may enhance performance (Mandengue *et al,* 1996).

Table 1.1 The most common physiological changes due to pre-exercise warm-up activities	
↑ Thermoregulatory responses during exercise	Torii *et al* (1996)
↑ Blood supply by vasodilatation	Karvonen, (1978)
↑ Heart rate and blood pressure	Barnard *et al* (1973)
↓ Affinity of haemoglobin for oxygen	Barcroft & King, (1909)
↑ Oxygen release from myoglobin	deVries, (1994)
↑ Mechanical efficiency	Hill, (1927)
↓ Time for redistribution of cardiac output	Karvonen, (1978)
↓ Reaction time	Kleitman *et al* (1938)
↑ Maximum Oxygen uptake	Yamaguchi, (1967)
↑ Hormonal mobilization	Caralis *et al* (1977)
↑ Joint range of motion	Lehmann *et al* (1970)
↑ Speed of nerve impulse	Hill, (1927)
↑ Muscle contractility	Bergh, (1980)
↑ Cardiorespiratory kinetics	Ingjer & Stromme,(1979)
↑ Neuromuscular co-ordination	Zatsiorsky, (1995)
↓ Bronchoconstriction in exercise-induced asthma	McKenzie *et al* (1994)
↓ Cardiac Ischaemia	Barnard *et al* (1973)
↓ Feelings of muscular soreness	Rodenburg *et al* (1994)
↑ Rate of chemical (metabolic) reactions	Bergh, (1980)

(↑ indicates 'increases' & ↓ indicates 'decreases'

1-4.3 Intensity, Type and Duration of Warm-up

Warm-up methods, when and if used, are more frequently based on the trial and error experience of the athlete or trainer, rather than on scientific study and advice. This is the case even when a relatively large number of performers have adopted warm-up practices. For example, Koutedakis *et al,* (1997) reported that, despite the fact that the majority of professional dancers are involved in warm-up routines,

only a few execute the routines according to scientific evidence. This may be partly due to the fact that the available literature is not conclusive regarding key parameters such as intensity and duration of warm-up.

Mitchell and Huston (1993), studied the influence of differing warm-up intensities upon swimming performance. The results indicated that varying the intensity of the warm-up protocol (70% to 110% of $\dot{V}_{O_2max}$) brings about no beneficial effects on performance. However, the design of the higher intensity warm-up did not take into account the accumulation of lactate, which might have affected the subsequent performance. Furthermore, Houmard *et al,* (1991) failed to show a significant improvement with event-specific warm-up intensity, but they confirmed that a warm-up consisting of mild-intensity, long-duration exercise is beneficial compared to no warm-up. In contrast, Zintl (1990) suggested that the technique of an activity should be rehearsed at the same intensity that will be encountered in the competition, but only in intervals that will not incur fatigue. In conclusion, varying the intensity and duration of warm up does not appear to lead to a significant alteration of $\dot{V}_{O_2}$ (Teubes *et al,* 1992).

Warm-up has particular significance for the asthmatic athlete. McKenzie *et al* (1994) found that a continuous warm-up of 15 min at 60 % of $\dot{V}_{O_2max}$ can significantly decrease post-exercise bronchoconstriction in moderately trained asthmatic athletes. It is thought that moderate exercises may actually cause bronchodilation, as an acute response of the lungs. Training appears to reduce the bronchial narrowing response and increases the bronchodilator response. In a study of 14 elite athletes with asthma, Todaro (1996) found evidence for bronchodilation post-exercise.

It has been known for some time that athletes with exercise-induced asthma have a refractory period after exercise, during which further exercise causes little bronchoconstriction (Weiler, 1996). This refractory period can last 1 - 4 hours, but

it may not occur in all individuals. The asthmatic athlete can make use of this by exercising at a level that does not induce bronchoconstriction for about 15 minutes before a performance. The mechanism(s) responsible for the refractory period is not fully understood.

The characteristics of warm-up such as intensity, duration, intervals between intensive exercise bouts and overall execution reflect on the effectiveness of each routine. Sen *et al* (1992) reported that an optimal combination of warm-up intensity with the intermission length allowed skiers to reach their maximal anaerobic power during laboratory assessments. It was suggested that the intensity of 50% of maximum heart rate was appropriate when skiing competition immediately follows warm-up preparations. When warm-up routines are completed five or more minutes prior to competition, then an intensity of 70% of maximum heart rate was suggested (Sen *et al,* 1992).

Even though body temperature increases can be induced by both passive and active warm-up protocols, there is a suggestion that the effect on performance varies. A study which examined the effects of a 45-min hot water immersion (44°C) prior to exercise, demonstrated no significant changes on selected isokinetic or isometric parameters such as knee extension peak torque, angle of peak torque, time to peak torque, average power, and isometric force (Stanley *et al,* 1994). The limited effectiveness of passive warm-up upon whole body performance suggests that a mere increase in body temperature is not the only responsible mechanism for the warm-up related performance enhancement (Astrand & Rodahl, 1987)

In competitive settings, warm-up activities also help to rehearse the skill component of the activity, focus on the upcoming event, and optimise the psychological component of competition. It has been suggested that pre-competitive preparation is experienced as a process resulting in optimal readiness

(Keating & Hogg, 1995), and that this process may comprise at least three phases: getting the body ready (physiological), getting a feel (psychophysiological), and getting the mind ready (psychological). In fact, some authors have felt that the period of preparation has a greater importance for maintaining the athlete in a state of optimal attentiveness and vigilance before the effort, rather than influencing the adaptation of the circulatory and respiratory systems (Lefebvre & De Bruyn, 1985; De Bruyn, 1990).

According to anecdotal evidence, warm-up routines prior to a rowing competition consist of both a general and specific warm-up during which both the physiological and psychological components of warm-up are addressed. A short period of general activity and stretching routines on-land comprises the general warm-up. On the water, a period of low intensity complements the general warm-up and prepares the peripheral muscles for the specific warm-up. During the specific warm-up intervals of high-intensity rowing are performed in an attempt to simulate the co-ordination and the recruitment level of the competitive movement pattern. However, no previous studies have investigated the effectiveness of such practice upon subsequent rowing performance .

1-4.4 Benefits for Long-term Performance

The recent work of Kesavachandran *et al* (1997) is in agreement with the view of Yamaguchi (1967) that warm-up increases vital capacity and decreases the minute ventilation for the same absolute workloads in preadolescent athletes. Similarly, warm-up has been shown to improve aerobic performances in both athletic and non-athletic children (Luehnenschloss & Niklass, 1988). Also, the study by Tomai *et al* (1996) supports the conclusions of Barnard *et al* (1973) on the effects of warm-up in delaying cardiac ischaemia.

A study with a 10-min cycling bout as warm-up, at a power output corresponding to the individuals' anaerobic threshold, showed significantly higher anaerobic

threshold values during a subsequent test compared to those of the control group (Black *et al,* 1984). It was concluded that cycling warm-up exercise can increase the sub-threshold oxygen uptake ($\dot{V}_{O_2}$) response. On the same note, Yoshida *et al* (1995) indicated that, at the onset of exercise, $\dot{V}_{O_2}$ kinetics are affected by the metabolic status of the active muscle. This finding suggests that $\dot{V}_{O_2}$ kinetics can be effectively influenced by prior warm-up activity of the muscle. According to Gerbino *et al* (1996) the faster $\dot{V}_{O_2}$ rise could be attributed to: a) an improved perfusion of the exercising muscles consequent to a vasodilatation effect of the acidaemia caused by a prior high-intensity exercise, and b) an acidaemia-induced Bohr shift of the haemoglobin dissociation curve leading to an improved O_2 diffusion gradient between the capillary blood and the mitochondria of the exercising muscle.

More recently, Koppo *et al* (1998) who investigated the $\dot{V}_{O_2}$ kinetics during high intensity cycling exercise preceded by high-intensity arm or cycling exercise, suggested that the faster $\dot{V}_{O_2}$ kinetics cannot be explained only by the effects of the induced acidaemia but other mechanisms related to the exercising musculature may also be involved. The same researchers also added that the total amount of prior work performed has an effect on $\dot{V}_{O_2}$ kinetics regardless of the chosen intensity (Koppo *et al,* 1998). Finally, Dempsey *et al* (1998) suggested that the exercise induced arterial hypoxemia observed during maximal exercise was decreased following prior preparatory activity.

These data suggests that warm-up can have a favorable impact on both the circulatory and respiratory systems. Consequently, long-term endurance performance may be enhanced.

1-4.5 Benefits for Short-term Performance

The suggestion of earlier studies (Karvonen, 1978; Bergh, 1980) that warm-up exercises are particularly beneficial in anaerobic physical efforts has been further supported by more recent studies. For example, the effects of a variety of warm-up routines on peak power (PP) and work capacity (WC) were studied by McKenna *et al* (1987). Using an air-braked cycle ergometer, these authors showed that warm-up significantly improved both PP and WC. However, an inverse relationship between warm-up intensity and short-term power output was observed by Sargeant and Dolan (1987) when warm-up activities exceeded 60% of the individuals' $\dot{V}_{O_2}$ max.

Following a maximal sprint cycle-ergometer test, blood lactate accumulation was decreased by approximately 50% when blood lactate levels were pre-elevated by arm-crank exercise (Bogdanis *et al,* 1994). It was speculated that this effect was caused by the reduced lactate efflux from the muscle rather than an improved lactate removal induced by the preliminary activity. Similarly, a standardised 200-m front crawl sprint swim was used to evaluate the effects of a variety of warm-up exercises – consisting of a 400-m front crawl swim (82% $\dot{V}_{O_2max}$), 400-m flutter kicking (45% $\dot{V}_{O_2max}$), and 4x50-m front crawl sprints (111% $\dot{V}_{O_2max}$) – on lactate levels (Robergs *et al,* 1990). Even though the warm-up resulted in increased pre-trial lactate concentrations, no further rise during the sprint swim was observed. These results indicate that selected warm-up exercise can minimise the disturbance in blood acid-base equilibrium during 2-min of intense swimming. It was proposed that the acid-base differences resulted from increased oxidative energy metabolism and a subsequent reduction in lactate and CO_2 production. However, when the effects of warm-up on glycogen degradation and energy metabolism during intense cycling exercise were studied, no statistical differences

were found between the 'warm-up' group and their controls, suggesting that warm-up does not have a direct effect on lactate production per se (Robergs *et al,* 1991).

In conclusion, there is good evidence suggesting that warm-up activities do improve short endurance performance by minimising the detrimental effects that exercise metabolites have on homeostasis and work capacity.

1-4.6 Benefits for Strength and Power Performance

Maximal strength, speed-strength and strength-endurance are the most common categories of strength, which are determined by the concerted activity of many muscles. It has been suggested that this intermuscular co-ordination should be optimised during warm-up by rehearsing the motor pattern of the event to follow (Zatsiorsky, 1995).

Guellich & Schmidtbleicher (1996) found that maximal volitional contractions, carried out during warm-up, could lead to a considerable increase in power performances of the lower extremities in athletic sprint and jumping events, as well as performance of the upper extremities in throwing events.

Comparing passive stretching and active warm-up (e.g. 10-min jogging), reaction time and force production were improved following the latter (Rosenbaum & Hennig, 1997). Similarly, the changes observed after a combined stretching and light-run protocol had a more pronounced improvement in force development (3%) and decrease (6%) in EMG activity, which can be viewed as performance-enhancing effects, than a stretching only treatment. (Rosenbaum & Hennig, 1985). It was noted that changes in the force characteristics such as reductions in the peak force, the force rise rate, the half relaxation rate, the EMG amplitudes and integrals and increases in EMG latencies observed after the stretching treatment indicate improved muscle compliance that might reduce the risk of injury.

Also, previous bouts of tetani have been shown to augment the subsequent tetanic force in isolated skeletal muscle fibres (Bruton *et al,* 1996). This potentiation, which lasted for at least 15 min, was thought to be due to a reduction in inorganic phosphate and may be a component of the physiological phenomena observed during warm-up.

Muscle power (P=W/t) is the explosive aspect of strength (often called "fast strength"). It is the functional application of both speed and strength, and is a key component in many human movements. A study by Rademaker (1996) found that a 45-min hot water immersion, which increased the muscle temperature by about 3°C, can have a significant increase in the power output at high velocities of human locomotion. The same authors also suggested the power output of type I fibres to be more sensitive to changes in muscle temperature than the output of type II fibres of human subjects.

In a study of baseball, DeRenne *et al* (1992) have shown that warming-up with an implement weighing within 10% of the bat's standard weight produces the highest bat swing velocities. In fact, a trend of decreasing velocity was found following warm-up, the more the weight of the bat deviated from the standard weight bat. However, this is in contrast with the findings of Bramford (1985) which suggest that using heavier equipment for warm-up can improve performance in competition. Perhaps, such results reflect the psychological advantage that is created by the sensation of overcoming greater loads.

In summary, the data presented are supporting the contention that warm-up may be beneficial to performances requiring strength and power.

1-4.7 Conclusions

In long-term endurance events, the functional status of the peripheral musculature is usually neglected by the athletes in favor of the more centrally oriented adaptations, brought about by temperature increases. Similarly, there is good evidence suggesting that warm-up activities have a positive effect on short-term performance. Temperature increases have also a positive effect on flexibility and range of movement with subsequent increases in mechanical efficiency, while the use of warm-up exercises prior to performance can reduce the chance of exercise-induced asthma occurring. Further research with elite performers will give us more insights into the optimal dose-response relationships for given sports. The studies presented in this thesis will aim to a) investigate whether the function of the respiratory muscles is improved following a specific respiratory warm-up protocol in a fashion similar to that observed in locomotory muscles, b) assess the effect of such intervention upon rowing performance and c) compare the effectiveness of different warm-up protocols upon rowing performance.

1-5. Summary

As described in the foregoing, respiratory limitations of exercise performance may arise in terms of gas exchange, respiratory mechanics, energetics of the respiratory muscles, or because of the development of respiratory muscle fatigue. Previous studies have suggested that respiratory muscle training can decrease exercise induced respiratory fatigue and associated dyspnea. Consequently, exercise performance may be improved in both patient and healthy populations.

During rowing the combination of the entrained breathing pattern, the mechanical limitations of the pulmonary system and the additional static supportive work for the upper body, place high demands upon the respiratory muscles. These demands predispose the respiratory muscles to fatigue despite the high fitness levels observed in rowers. Due to the various implications that respiratory muscle fatigue can have upon rowing performance, the aim of this thesis will be: a) to investigate the incidence of respiratory muscle fatigue during rowing, b) to assess the influence of IMT and specific inspiratory muscle warm-up upon inspiratory muscle fatigue and c) to evaluate the effect of such interventions upon rowing performance.

Our hypothesis is that inspiratory muscle loading (acute or chronic) can have a favorable effect upon rowing performance.

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Chapter Two

The Influence of Prior Activity Upon Inspiratory Muscle Strength in Rowers and Non-rowers.

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2-1. ABSTRACT

The aim of this study was to investigate whether a 'warm-up' phenomenon exists for the inspiratory muscles and, under this assumption, to compare the benefits of a whole body warm-up and a specific respiratory warm-up.

Eleven club level rowers performed a rowing warm-up and twelve university students performed a general cycling warm-up. Both groups also performed a specific respiratory warm-up. Inspiratory muscle strength (Mueller manoeuvre) and lung function (flow-volume loops) were measured before and after the three conditions. Isokinetic strength during knee extension was measured before and after the rowing warm-up.

The two whole body warm-up protocols had no effect on inspiratory muscle strength or any lung function parameter despite the significant (3.8 ± SD 1.4 %; $p < 0.05$) increase in peak torque that the rowing warm-up elicited. The respiratory warm-up induced an increase in inspiratory mouth pressure (8.5 ± 1.8 %; $p < 0.0001$) but not in any other lung function parameter. Following the rowing incremental test to exhaustion maximum inspiratory pressure decreased by 7.0 ± 2.0 %, which was interpreted as an indication of respiratory muscle fatigue.

These data suggest that the inspiratory muscle strength can be enhanced with preliminary activity, a phenomenon similar to the one known to exist for other skeletal muscles. In addition, a specific respiratory warm-up is more effective in this respect than whole body protocols.

2-2. INTRODUCTION

Warm-up may be defined as any preliminary activity that is used to enhance physical performance and to prevent sports-related injuries. There are various types of warm-up techniques that competitors use to prepare for their event. The most widely used methods are classified as *passive, general* and *specific warm-up* (Shellock & Prentice, 1985).

Competitive rowing is considered to be one of the most demanding sports, as rowers work near their maximal physical capacities and recruit a very large muscle mass. Open class rowers generate amongst the highest values of any athletes in selected physical fitness parameters, including those related to cardiorespiratory and muscular function (Koutedakis *et al,* 1990). Warm-up is an integral part of the preparation before the start of the race.

Most general warm-up protocols are of moderate intensity and characterised by a low ventilatory demand (Karvonen, 1992). In competitive rowing, however, a higher intensity specific warm-up usually follows the general warm-up in an attempt to practise the racing pace (Grosser, 1991). The higher intensity of the specific warm-up, among other peripheral adaptations, elicits an elevated ventilatory response that may prepare the respiratory muscles for the demanding entrained breathing of rowing (Mahler *et al,* 1991; Steinacker *et al,* 1993). The effect of warm-up upon locomotor muscle strength is well documented (Hill, 1927; Binkhorst *et al,* 1977; Bergh & Ekblom, 1979; de Vries, 1980) but very little scientific attention has been directed towards the effect of warm-up on pulmonary function and specifically inspiratory muscle strength.

The present study addressed the following questions: a) Does a whole body warm-up influence inspiratory muscle strength? b) Does a specific respiratory warm-up affect the inspiratory muscle strength? Accordingly, we compared the

effects of 3 warm-up protocols, a general cycling warm-up, a rowing warm-up and a specific respiratory warm-up. Our hypothesis was that the inspiratory muscles will exhibit an improvement in performance similar to that observed in other skeletal muscles following at least one of the 3 warm-up protocols.

2-3. METHODS

2-3.1 Subjects

Twenty-three subjects participated after giving informed written consent to the study that was approved by the local Ethics Committee. Twelve Sport Science students formed the 'non-rowing' group and eleven club level rowers formed the 'rowing' group (table 2.1). One of the subjects was removed from the study because he developed a respiratory tract infection within two weeks of the data collection, a condition known to have potential effects on respiratory muscle strength (Mier-Jedrzejowicz *et al,* 1988).

Table 2.1 Characteristics of the two groups.

Mean ± SD	Non-rowing (n=12)	Rowing(n=11)
Age (yr)	20 ± 1	20 ± 2
Height (cm)	175 ± 8	180 ± 6
Weight (kg)	70 ±11	74 ± 8
FVC (L)	5.4 ± 0.9	5.3 ± 0.6
FEV_1 (L)	4.6 ± 0.6	4.5 ± 0.5
FEV_1/FVC (%)	86 ± 8	87.2 ± 7
$\dot{V}_{O_2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	47 ± 7*	58 ± 7**

* Measured during cycling, ** measured during rowing.

2-3.2 Procedure

Before data collection, all subjects visited the lab on two occasions to be familiarised with mouth pressure measurements and flow volume manoeuvres. Following familiarization, two additional visits took place for collection of reproducibility data. Subsequently, both groups performed an incremental test to volitional exhaustion, a whole body warm-up and a specific respiratory warm-up, which took place in three separate occasions. The non-rowing group, which used a cycle ergometer for the incremental test and the whole body warm-up, performed a general warm-up while the rowing group, which used a rowing ergometer for the respective exercise sessions, performed a rowing warm-up consisting of a general and a sport specific warm-up. The specific respiratory warm-up was performed using a pressure threshold inspiratory muscle training device. Maximum mouth pressures and pulmonary function were assessed before and after every treatment condition. Additionally, as an index of the warm-up effect on the peripheral musculature, isokinetic strength of the quadriceps was measured before and after the rowing warm-up.

2-3.3 Maximum Inspiratory Pressures (MIP)

MIP is commonly used to measure inspiratory muscle strength. It reflects the force-generating capacity of the combined inspiratory muscles during a brief, quasi-static contraction (Mueller manoeuvre)(Larson *et al,* 1993). MIP was recorded using a portable hand held mouth pressure meter, (Precision Medical, UK). This device has been shown to measure inspiratory efforts accurately and reliably (Hamnegard *et al,* 1994). A minimum of five and a maximum of nine technically satisfactory measurements were conducted and the highest of three measurements with 5% variability or within 5 cm H_2O difference, was defined as maximum (Wen *et al,* 1997). The initial length of the inspiratory muscles was controlled by initiating each effort from residual volume (RV). This procedure was adopted because, from our experience, RV is more reproducible than functional residual capacity (FRC). Subjects were instructed to take their time and to slowly

empty their lungs to RV, thereby avoiding problems associated with variability in lung volumes. All manoeuvres were performed in the upright standing position and verbal encouragement was given to help the subjects perform maximally.

2-3.4 Static Spirometry

Pulmonary function was assessed with a Vitalograph 2120 portable spirometer (Vitalograph Ltd., Buckingham, England), which was calibrated prior to each testing session using a 3 litre calibration syringe (Hans Rudolph inc., Kansas, USA). Following familiarisation, the best of three manoeuvres were recorded. Forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), percentage expired (i.e. 100 x FEV_1/FVC)(FEV_1 %) and peak inspiratory flow rate (PIFR) were the parameters recorded before and after every treatment condition.

2-3.5 Incremental Test to Exhaustion (Peak$\dot{V}_{O_2}$)

The non-rowing group performed a continuous incremental protocol to volitional exhaustion on an Excalibur Sport V2.0 electromagnetically braked cycle ergometer. The work rate was increased every fifteen seconds and was designed to elicit maximal oxygen uptake (peak $\dot{V}_{O_2}$) within ten to twelve minutes. The test was terminated at volitional exhaustion or when the subject failed to maintain a pedalling frequency higher than 50 rpm.

The rowing group performed an incremental test to volitional exhaustion on a wind-resistance braked rowing ergometer (Concept II, model c, Morrisville, USA) starting at an individually chosen light work intensity and increasing the workrate by 50 W every 3 minutes. The wind damper was at the fourth setting. Power was calculated from acceleration of the flywheel and displayed on a monitor. Maximal power (Pmax) was calculated as

$$P_{max} = P_{n-1}+([[P_n- P_{n-1}] \cdot t_n] /180)$$

with P_n = power of the maximum stage, P_{n-1} = power of the stage before, and t_n= time of work of the maximum stage in seconds (Lormes *et al,* 1993).

Breath by breath gas analysis was made with an MGA 2000 Mass Spectrometer (Airspec Ltd., Kent, UK) in conjunction with an ultrasonic phase-shift flowmeter (Birmingham Flowmetrics, Birmingham, UK). Data processing was performed on-line (Labview 3, National Instruments, Austin TX, USA) on a Powermac 7100/80 (Macintosh Ltd., USA). Calibration of the flowmeter was performed before each test using a 1 litre calibration syringe (PK Morgan Ltd., Kent, UK). The heart rate was telemetrically monitored with Polar Accurex Plus heart rate monitor (Polar Electro, Finland).

2-3.6 General Warm-Up

Twenty min of cycling was performed on the same cycle ergometer as in the incremental test. The first 10 min were performed at 30% of peak work rate (WRpeak), the next 5 min at 35% and the final 5 min at 40%. Pedalling frequency was maintained between 70-80 rpm. This modest-intensity protocol was intended to assimilate the general warm-up preceding the sport specific warm-up. Breath by breath gas exchange analysis and heart rate data were collected as during the peak $\dot{V}_{O_2}$ test. Post warm-up measurements were made within two minutes of completion.

2-3.7 Rowing Warm-Up

The protocol was designed to mimic as closely as possible the routine that is usually adopted in preparation for a rowing race. Five minutes of very light jogging on the treadmill, at a heart rate of 110-130b/min, were followed by 10 minutes of stretching. Subsequently, 12 minutes rowing of gradually increasing intensity were performed during which the heart rate increased from 148 (±2) to 178 (±2) b/min. The increase in intensity was achieved primarily by increasing the stroke rate.

Then, 5 sprints with increasing stroke rate and power output were performed. Between each sprint there was an active rest interval of light paddling which lasted approximately 2 minutes. At the end of the sprints, the rower rested for approximately 5-7 minutes before any further measurements were made. This rest interval was designed to simulate the small pause between the end of the warm-up and the start of the race. Details of the structure of the Rowing Warm-up can be seen in table 2.2. Breath by breath gas analysis and heart rate data were collected throughout.

Table 2. 2 Description of the Rowing Warm-Up on the rowing ergometer.

Warm-Up (time)	Stroke rate/min @	Percent Power Max (% Pmax)
1x12min (4-4-3-1)	18-20-22-24	50-60-70-75
2x30s	26-28	94.7(±3.7) -103.6(±2.6)
2x45s	28	108.9(±2.9) -115 (±2.6)
1 min	30-32	132.2 (± 5.0)

%Pmax = percentage of maximum power output achieved during the 6 min all-out effort

2-3.8 Respiratory Warm-Up

Two sets of 30 breaths were performed using POWERbreath® inspiratory muscle trainer (IMT Technologies Ltd., Birmingham, UK) at 40% of the MIP measured previously. Between the two sets there was a short rest interval while an intermediate MIP measurement was made. Forty percent of maximum capacity has been suggested to approximate the upper loading limit before fatigue of the diaphragm occurs (Roussos & Macklem, 1977). POWERbreath® is a pressure-threshold device which requires continuous application of inspiratory pressure throughout inspiration in order for the inspiratory regulating valve to remain open. As with the maximal inspiratory pressures, subjects were instructed to initiate every breath from RV. They continued the inspiratory effort up to the lung volume

where the inspiratory capacity for the given resistance limited further excursion of the thorax. Powerful execution of the manoeuvres was encouraged to ensure maximal voluntary output for the given loading conditions. Because of the increased tidal volume, a decreased but spontaneous breathing frequency was adopted by the subjects in order to avoid hyperventilation.

2-3.9 Isokinetic Strength

Dynamic isokinetic strength was measured before and after the rowing warm-up. Peak Torque (Nm) and angle (degrees) of peak torque was measured during a concentric knee extension of the dominant leg on a Cybex Norm isokinetic dynamometer (Cybex International, Inc. Ronkonkona, New York, USA). A relatively slow speed of 60°/sec was chosen to approximate the slow velocity encountered in rowing. All subjects had at least two practice trials on previous occasions for familiarisation with the nature of the dynamometer and the specific testing velocity. On the test day three practice trials with a light effort preceded the three maximum efforts from which the best value was taken for further analyses.

2-3.10 Statistical Analyses

Student's t-test for paired samples was used to compare differences between the MIP values before and after the two whole body warm-up protocols. ANOVA with repeated measures and Scheffé post-hoc test was used to assess differences in the RespWU. Values of $P < 0.05$ were considered statistically significant. Data points were means (± SE) unless otherwise stated.

2-4 RESULTS

2-4.1 Rowing Warm-Up and General Warm-Up Characteristics

Compared with the peak $\dot{V}_{O_2}$ test the Rowing Warm-up and the General Warm-up elicited a ventilatory response with the characteristics shown in table 2.3.

Table 2.3. Data obtained from the General and the Rowing warm-up expressed as percent peak values observed during the peak $\dot{V}_{O_2}$ test (Data for the Rowing warm-up is from the 12 minutes continuous rowing phase).

Mean (± SE)	General	Rowing
$\dot{V}_E$%	40.1 (± 6.9)	70.1 (± 2.6)
$\dot{V}_{O_2}$%	62.3 (± 9.5)	80.5 (± 2.4)
f_C%	71.2 (± 3.2)	90.1 (± 1.0)
V_T%	88.1 (± 12.6)	88.2 (± 1.7)
f_R%	52.7 (± 5.8)	76.6 (± 3.1)
PIFR %	47.4 (± 9.6)	65.1 (± 1.3)

$\dot{V}_E$= minute ventilation, V_T= tidal volume (inspired), f_R = frequency of breathing, PIFR = peak inspiratory flow rate, f_C = cardiac frequency.

2-4.2 Isokinetic Strength

The peak torque of the leg extension increased significantly after the rowing warm-up by 3.8 (± 1.4)% ($P < 0.05$). The angle of peak torque increased by 2.8 (± 3.1)% but this increase was not significant.

2-4.3 MIP

Test - Retest Reproducibility of MIP

The two baseline measurements, i.e., before the whole body and respiratory warm-ups, permitted a test-retest assessment of MIP. For the comparison of baseline MIP values the data of both groups were pooled. The mean baseline MIP values of the whole body warm-up protocols and the respiratory warm-up were not significantly different, the mean difference being less than 5 cm H_2O. The mean (± SE) coefficient of variation (CV = 100% x SD/mean) for the baseline MIP measured on the two occasions was 4.65 (± 0.76) %. Additional analysis using the Bland-Altman plot (6)(fig. 2.1), revealed a repeatability coefficient of 26.6 cm H_2O.

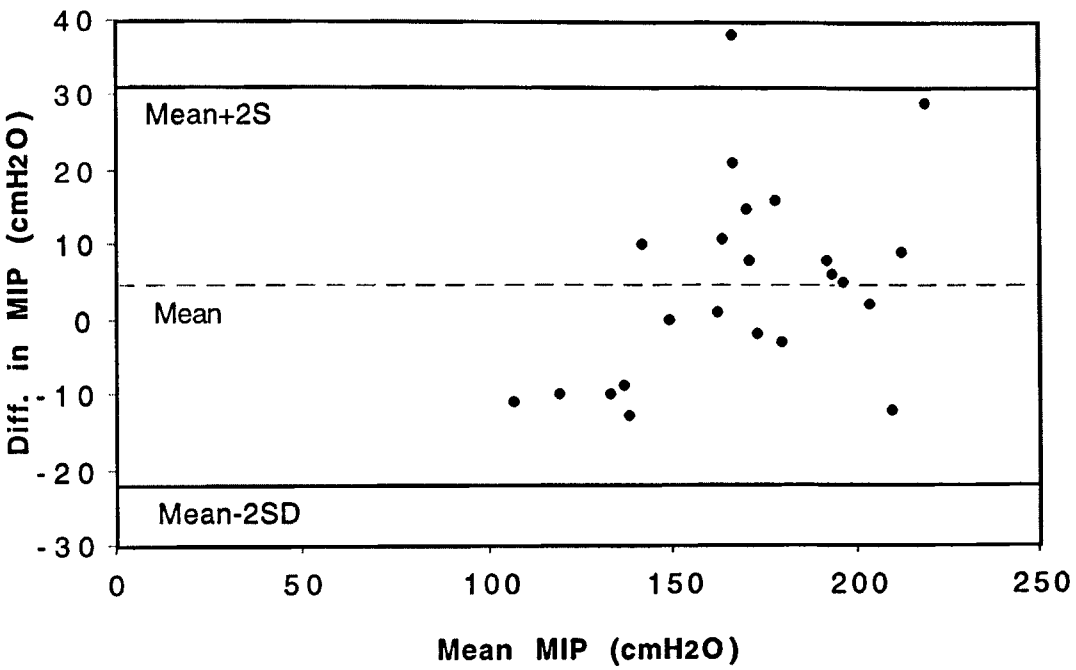

Fig 2.1 Bland- Altman plot for reproducibility of baseline MIP between whole body warm-up protocols and Respiratory Warm-up.

Influence of Maximal testing Upon MIP

Immediately after the incremental cycling (peak $\dot{V}_{O_2}$) test MIP decreased by 2.2 ± 3.0 % from the baseline; this difference was not significant. After the incremental rowing (peak $\dot{V}_{O_2}$) test MIP decreased by 7.0 ± 2.0 % ($P < 0.01$).

Influence of Whole Body Warm-Up on MIP

For the comparison between whole body warm-up protocols the two groups have been analysed separately. After the General Warm-up, MIP increased from a baseline of 171.4 (± 9.0) cm H_2O to 178.8 (± 12.6) cm H_2O, a mean (± SE) percent increase of 3.4 (± 2.5) %; this difference was not significant ($p > 0.05$). After the Rowing Warm-up, baseline MIP increased from a mean of 161.1(± 7.5) cm H_2O to

162.8 (± 10.7), a mean (± SE) percent increase of 0.3 (± 3.2)% which again was not significant (p >0.05).

Influence of Respiratory Warm-Up on MIP

For the comparison of MIP values before and after the Respiratory Warm-Up which was common for both groups the data were pooled. The Respiratory Warm-Up induced a significant increase in MIP from a mean baseline of 171.2 (± 7.0) cm H_2O to 178.1 (± 6.8) cm H_2O after 30 breaths, a 4.5 ± 1.1 % increase (P<0.001). After 60 breaths the mean MIP increased further to 184.2 (± 6.4) cm H_2O, an additional significant increase of 3.8 ± 1.3 % (p< 0.01). The total increase from baseline was 8.5 ± 1.8 % (P<0.0001).

2-4.4 Lung Function

There were no significant changes in the parameters measured other than MIP. Pulmonary function data obtained after the General, Rowing and Respiratory Warm-ups are summarised in table 2.4.

Table 2.4. Mean (SE) percent changes between baseline and the three warm-up protocols. Results shown under Respiratory warm-up are pooled data for both groups.

	General (n=12)	Rowing (n=11)	Respiratory (n=23)
MIP %	3.4 (±2.5)	0.3 (±3.2)	8.5 (±1.8)*
FVC %	1.9 (±2.4)	-1.0 (±1.4)	1.0 (±1.2)
FEV_1 %	-1.0 (±1.3)	0.4 (±0.9)	0.4 (±0.9)
FEV_1/FVC %	-1.3 (±1.5)	1.4 (±1.5)	1.4 (±1.5)
PIFR %	-1.2 (±2.3)	0.3 (±3.1)	1.7 (±2.1)

MIP = maximum inspiratory pressure, PIFR = peak inspiratory flow rate,
*Denotes significance (p<0.0001)

2-4.5 Prediction of Warm-Up Effect

Post-Respiratory Warm-Up MIP was significantly correlated with the baseline MIP ($p<0.001$) and this relationship can be described by the two linear models on table 2.5, derived from data taken after the two sets of 30 breaths of the Respiratory Warm-up.

Table 2.5. Predictive equations for MIP.

Respiratory Warm-up	R^2	Regression equation
30 breaths	0.9409	y = 0.9344 x + 18.099
60 breaths	0.8667	y = 0.8506 x + 38.539

y = New MIP, x = baseline MIP.

2-5 DISCUSSION

The main finding of this study was that MIP increased significantly following the Respiratory Warm-up but not following the two whole body warm-up protocols. This phenomenon, which emerges with at least 30 breaths using POWERbreath®, raises the possibility that the respiratory system may have different warm-up requirements (threshold) than the locomotor system.

Emphasis was given to the methodological issues related with the Mueller manoeuvre. The variability in MIP between baselines is in agreement with previous reports on test-retest reproducibility (Larson *et al,* 1993). The mean coefficient of variation, which was smaller than reported previously (Black & Hyatt, 1969; Wilson *et al,* 1984; Aldrich & Spiro, 1995), as well as the coefficient of repeatability from the Bland-Altman plot, which is in agreement with the study of Maillard *et al* (1998), suggest that the task learning effect was expressed and reliable baselines were established.

Another interesting observation was that following the incremental rowing test to exhaustion MIP decreased, whilst no significant changes occurred after the incremental cycling protocol. These data are suggestive of respiratory muscle

fatigue and are in agreement with previous reports of the effect of exhausting exercise upon respiratory muscle function (Mahler & Loke, 1981; Hill *et al,* 1991; Johnson *et al,* 1996; McConnell *et al,* 1997). During rowing, thoracic muscles are responsible not only for the act of breathing but also for the stabilisation of the thorax (Dal Monte & Komor, 1989). This additional role of respiratory muscles in the locomotive work of rowing might be the reason for the development of inspiratory muscle fatigue in such a short time compared with longer exercise durations reported previously. Even though the entrained breathing observed in rowing is suggestive of a possible scenario for respiratory muscle fatigue no study has reported it previously. Clearly, more work needs to be done in the breathing requirements of rowing to understand the functional significance of these findings.

The precise mechanism(s) responsible for the increase in MIP following the Respiratory Warm-up can not be identified easily. A skeletal muscle warm-up has been reported to have an effect on maximum isometric force when the change in the muscle temperature is substantial (Bergh & Ekblom, 1979; Ranatunga *et al,* 1987). However, since in the present study it was not possible to measure the temperature of the diaphragm or the intercostal muscles, we can only suggest that a temperature related effect, if any, was unlikely. This suggestion is justified under the assumption that the temperature of the diaphragm and the other inspiratory muscles is essentially equal to the core temperature because of their location.

Thus, by a process of elimination, an altered motor control hypothesis is suggested. It is possible that the intermuscular co-ordination between inspiratory and expiratory muscles is improved in a manner similar to the one identified for other skeletal muscles (Komi, 1992). Repeated performance of the specific recruitment pattern might decrease the degree of co-contraction known to exist between inspiratory and expiratory muscles at RV and consequently improve force generation.

The protocols used in the General Warm-up and the Rowing Warm-up, did not alter MIP. A possible explanation may be that, due to the modest ventilatory response elicited by the General Warm-up, the threshold required for the respiratory muscles 'warm-up' was not achieved. However, during the Rowing Warm-up the ventilatory response was more pronounced, as can be seen from table 3, but again MIP did not change. Comparing the breathing patterns of the two whole body warm-up protocols we notice that thoracic excursions were of similar magnitude. The elevated minute volume of the Rowing Warm-up was effected through increases in breathing frequency as expected. These sub-maximal unloaded breathing patterns, predominantly characterised by diaphragm participation, are different from the pattern of a relative chest wall muscle recruitment observed during the Mueller manoeuvre (Nava *et al,* 1993). Therefore, the recruitment pattern involved could be suggestive of a relative insensitivity of the Mueller manoeuvre to tension changes effected by diaphragm participation.

In contrast, during the Respiratory Warm-up the recruitment of the chest wall muscles is substantial as loading compensation enhances the inspiratory activity of the external intercostal muscles. Furthermore, deliberate inspiratory efforts tend to make greater use of inspiratory intercostal muscles of the chest wall than do spontaneous metabolically stimulated inspirations (Whitelaw & Feroah, 1989). It has often been observed in strength-training studies that increases in strength depend on how similar the strength test is to the actual training exercise in terms of muscle fibre length and type of contraction (Sale & MacDougall, 1981). Indeed, the recruitment pattern of the Mueller manoeuvre is more similar to the pattern of the Respiratory Warm-up than the pattern of the two whole body warm-up protocols.

The Rowing Warm-Up increased the peak torque measured during concentric knee extension and confirms its effectiveness as a preliminary activity. These data are also in agreement with previous reports on the beneficial effects of sport

specific warm-up (Karvonen, 1992; Shellock & Prentice, 1985). The fact that the rowing warm-up failed to enhance MIP suggests that the respiratory muscles may not be optimally prepared before the start of a rowing race. Additionally, the possibility of a discrepancy between the work intensity required, for an enhanced function of the respiratory muscles and the muscles of locomotion, is raised. An improved functional capacity of the inspiratory muscles, as a result of warm-up, may allow a decrease in recruitment requirements and minimise in doing so the sensation of breathlessness. Indeed, strong relationship between recruitment of the inspiratory muscles and the perception of dyspnea has been suggested (Killian & Jones, 1988). More work is needed to investigate the potential effect of this upon the perception of breathlessness and rowing performance.

Finally our data suggest that in the clinical and academic fields, studies that examine the function of the inspiratory muscles under different treatment conditions should account for a 'warm-up' effect on baseline MIP. Indeed, studies examining post-exercise inspiratory muscle fatigue might reveal that the degree of fatigue reported is larger than previously thought. Likewise, results from studies that have failed to observe the presence of fatigue may have done so because it was masked by the 'warm-up' effect.

2-6 CONCLUSIONS

A warm-up phenomenon, similar to the one present in locomotion, exists in the inspiratory muscles. This enhancement is more effectively elicited by specific inspiratory manoeuvres than by whole body warm-up protocols.

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Chapter Three

Prior Submaximal Respiratory Muscle Activity ('WarmUp') Enhances Maximum Inspiratory Pressure (PImax) and Improves Reliability of the Assessment/Measurement of PImax.

Part of this chapter have been accepted for publication in *Respiration.*

3-1 ABSTRACT

The variability of maximal inspiratory pressure (PImax) in response to repeated measurement affects its reliability; published studies have used between three and twenty PImax measurements in a single occasion. This study investigated the influence of a specific respiratory 'warm-up' upon the repeated measurement of inspiratory muscle strength and attempts to establish a procedure by which PImax can be assessed with maximum reliability using the smallest number of manoeuvres.

Fourteen healthy subjects, familiar with the Mueller manoeuvre, were studied. The influence of repeated testing on a single occasion was assessed using an 18 measurements protocol. Using a randomised cross-over design subjects performed the protocol, preceded by a specific respiratory warm-up (RWU) and on another occasion without any preliminary activity (Control). Comparisons were made amongst 'baseline' (best of the first 3 measurements), 'short' series (best of 7^{th} to 9^{th} measurement) and 'long' series (best of the last 3 measurements).

Under control conditions the mean increase ('baseline' vs. 'long' series) was 11.4 (5.8)%; following the RWU the increase (post RWU 'baseline' vs. 'long' series) was 3.2 (10.0)%. There were statistically significant differences between measurements made at all 3 protocol stages ('baseline', 'short' and 'long' series) under control conditions, but none following the RWU.

The present data suggest that a specific RWU may reduce the 'learning effect' during repeated PImax measurements which is one of the main contributors of the test's variability. The use of a RWU may provide a means of obtaining reliable values of PImax following just 3 measurements.

3-2 INTRODUCTION

Maximal inspiratory pressure (PImax) is a commonly used index of inspiratory muscle strength; it reflects the combined force-generating capacity of the inspiratory muscles during a brief, quasi-static contraction (Mueller manoeuvre)(Larson *et al,* 1993). The reliability of PImax has been questioned, particularly in the context of its variability in response to repeated measurement. Day to day fluctuations similar to those seen with other measures of strength have been reported to be $\pm$ 10 percent (Astrand & Rodahl, 1986). However, the variability relating to the number of manoeuvres that are performed can result in an underestimate of PImax of as much as 20 cmH_2O (Wen *et al,* 1997). Reports suggest that a learning effect can be significant in both short series (3-5 measurements) and longer series (up to 20 measurements) protocols (Fiz *et al,* 1989; Wen *et al,* 1997).

In a recent study, we have shown that prior activity of the inspiratory muscles, i.e., breathing against a modest threshold load using an inspiratory muscle trainer, induced a statistically significant increase in PImax (Volianitis *et al,* 1999).

Thus, the aims of the present study were to investigate the influence of a specific respiratory 'warm-up' upon the repeated measurement of inspiratory muscle strength and to attempt to establish a procedure by which PImax can be assessed with maximum reliability using the smallest number of manoeuvres. The influence of repeated testing was assessed on a single occasion using a protocol which consisted of a total of 18 maximal efforts.

3-3 METHODS

3-3.1 Subjects

Fourteen healthy subjects were studied following informed, written consent and local Ethics Committee approval. Their mean ± SD characteristics were: age 26 ± 3 yrs; weight 72 ± 9 kg; and height 1.80 ± 0.06 m. None reported any history of respiratory or neuromuscular disease. Subjects were removed from the study if they reported a respiratory tract infection within the two weeks of data collection because of the potential effects upon respiratory muscle strength (Mier-Jedrzejowicz *et al,* 1988).

3-3.2 Procedure

Subjects were experienced with the nature of the Mueller manoeuvre or had visited the laboratory at least twice for familiarisation prior to data collection. These two preliminary sessions were designed to allow for the initial learning effect and to assess test-retest reliability. Larson *et al* (1993) reported that performance plateaus between the 3rd and 4th test session. The familiarisation sessions consisted of the same protocol as in the actual data collection. The influence of repeated testing on a single occasion was assessed using a protocol consisting of a total of 18 measurements, performed in 6 sets of 3 efforts. One minute rest was allowed between individual measurements and a 3 min rest between sets to minimise the effects of fatigue.

Subjects performed the protocol under two conditions, in a randomised cross over design. On one occasion, the test protocol was preceded by a specific respiratory warm-up (RWU); on another occasion, the protocol was performed without any preliminary activity (Control). Both data collection sessions (Control and RWU) were performed at the same time of the day and within 5-7 days of each other.

3-3.3 Maximum Inspiratory Pressures

Peak maximum inspiratory pressure (P.PImax) and maximum inspiratory pressure averaged over 1 sec (PImax) were recorded using a portable hand held mouth pressure meter, (Morgan Medical, UK). This device, equipped with a flanged mouthpiece, has been demonstrated to measure inspiratory efforts accurately and reliably (Hamnegard *et al,* 1994). A small hole in the system was preventing closure of the glotis during inspiration and a noseclip was used in all efforts. The subjects were asked to sustain a maximal inspiratory effort for 2-3 sec.

The initial length of the inspiratory muscles was controlled by initiating each effort from residual volume (RV). Subjects were instructed to take their time and to slowly empty their lungs to RV. All manoeuvres were performed in the upright standing position and verbal encouragement was given to help the subjects perform maximally. The trials which did not represent the subjects' maximum effort, according to their subjective feeling, were discarded.

Comparisons were made according to the following definitions: 'baseline' series measurement was defined as the highest of the first 3 measurements. The 'short' series measurement was defined as the highest among the 7^{th} to the 9^{th} efforts (Wen *et al,* 1997). The 'long' series measurement was defined as the highest value amongst the last 3 of the 18 measurements.

3-3.4 Specific Respiratory Warm-Up (RWU)

Two sets of 30 breaths were performed with a POWERbreathe® inspiratory muscle trainer (IMT Technologies Ltd., Birmingham, UK), with mouth pressures being measured within 2 minutes of completion of each set. The pressure load was set at 40% of the PImax measured before the commencement of the protocol. This level of recruitment has been suggested to approximate the upper loading limit before fatigue of the diaphragm occurs (Roussos & Macklem, 1977).

POWERbreathe® is a pressure-threshold device which requires continuous application of inspiratory pressure throughout inspiration in order for the inspiratory regulating valve to remain open. Subjects were instructed to initiate each breath from RV and to continue the inspiratory effort up to the lung volume where the inspiratory muscle force output for the given load limited further excursion of the thorax. Because of the increased tidal volume, a decreased breathing frequency was adopted in order to avoid hyperventilation and the consequent hypocapnia.

3-3.5 Statistical Analyses

Student's t-test for paired samples was used to compare differences before and after the two conditions. The coefficient of variation (CV = 100% x SD/mean) and the coefficient of repeatability for agreement (Bland & Altman, 1986) was used to evaluate the within-sessions reproducibility of the baseline and final maximum values. One-way ANOVA with repeated measures and Scheffé post-hoc test was used to detect differences between the 'baseline', 'short' and 'long' series values. $P < 0.05$ was considered statistically significant.

3-4 RESULTS

3-4.1 Test-retest Reproducibility

The group mean ± SD 'baseline' and 'long' series PImax and P.PImax values measured under the two test conditions are summarised in Table 3.1 (note that the 'baseline' value presented here for RWU is the value recorded prior to the RWU).

Table 3.1 Mean (SD) values of PImax and P.PImax (kPa): difference between baseline series values without RWU and long series values with and without RWU.

	PImax		P.PImax	
	Baseline	Long	Baseline	Long
Control	13.5±2.4	14.7±2.8	13.5±2.4	15.9±2.8
RWU	13.5±2.4*	14.7±3.0	13.9±2.1*	15.7±2.9

Baseline: highest of the first 3 measurements, * recorded prior to the RWU; Long: highest of the last 3 measurements; PImax: Maximum inspiratory pressure averaged over 1s; PPImax: Peak maximum inspiratory pressure; RWU-Respiratory warm-up. Note: Baseline value for RWU in the value recorded prior to the RWU.

The mean (SE) coefficient of variation (CV = 100% x SD/mean) and the repeatability coefficient (Bland & Altman, 1986) of the 'baseline' and 'long' series PImax and P.PImax values for the two conditions are shown in Table 3.2.

Table 3.2 Reproducibility data for the 'baseline' and 'long'series measurements of PImax and P.PImax.

	PImax		P.PImax	
	Baseline	Long	Baseline	Long
CV (%)	8.4±3.7	5.3±3.1	8.2±4.7	5.6±3.7
CR (kPa)	3.7	2.5	3.7	3.0

CV-Coefficient of variation, mean (SD); CR-Coefficient of repeatability for agreement.

3-4.2 Response to the RWU

Following the RWU, there was a significant increase in the mean baseline values of both PImax and P.PImax ($P < 0.01$) which, in absolute values, was 1.4 ± 0.5 kPa (13.8 ± 5.6 cm H_2O) and 1.6 ± 0.6 kPa (15.9 ± 5.8 cm H_2O), or 10.1 ± 8.1% and 11.2 ± 9.0 %, respectively.

Response to repeated measurement

The development of PImax and P.PImax values during the 18 measurement protocol under both conditions is summarised in Table 3.3 and for PImax in Figure 3.1. Under control conditions the mean increase ('baseline' vs. 'long' series) was 11.4 (5.8)%; following the RWU the increase (post RWU 'baseline' vs. 'long' series) was 3.2 (10.0)%. There were statistically significant differences between measurements made at all 3 protocol stages ('baseline', 'short' and 'long' series) under control conditions, but none following the RWU.

Table 3.3 Mean ± SD development of PImax and P.PImax values (kPa) throughout the 18 measurement protocol for the two conditions (* P< 0.05, ** P< 0.01).

	PImax			P.PImax		
	Baseline	Short	Long	Baseline	Short	Long
Control	13.2±2.2	*13.8±2.7	**14.7±2.8	14.2±2.3	*15.0±2.8	**15.9±2.8
RWU	14.3±2.9	14.2±2.3	14.7±3.0	15.5±2.7	15.5±2.8	15.7±2.9

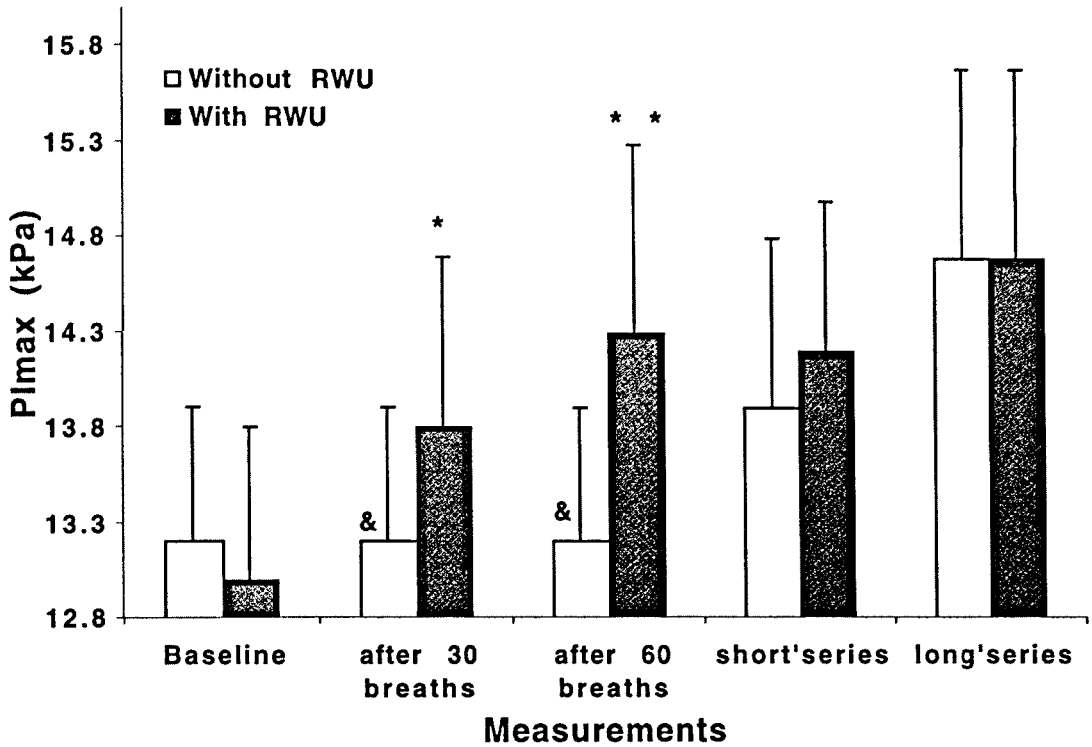


Fig 3.1 Development of Pimax during the Warm-up and the following 18 measurement protocol under both conditions. Comparisons are between RWU and control conditions. (* P< 0.05, ** P< 0.01) (&: baseline' values of the trial without RWU are repeated for comparison purposes).

During the 18 measurements without the RWU, PImax developed a plateau at the 8-10 measurement and a further stabilisation at the 17-18 measurement (fig. 3.2).

Fig 3.2 Development of PImax during the 18 measurement protocol without RWU.

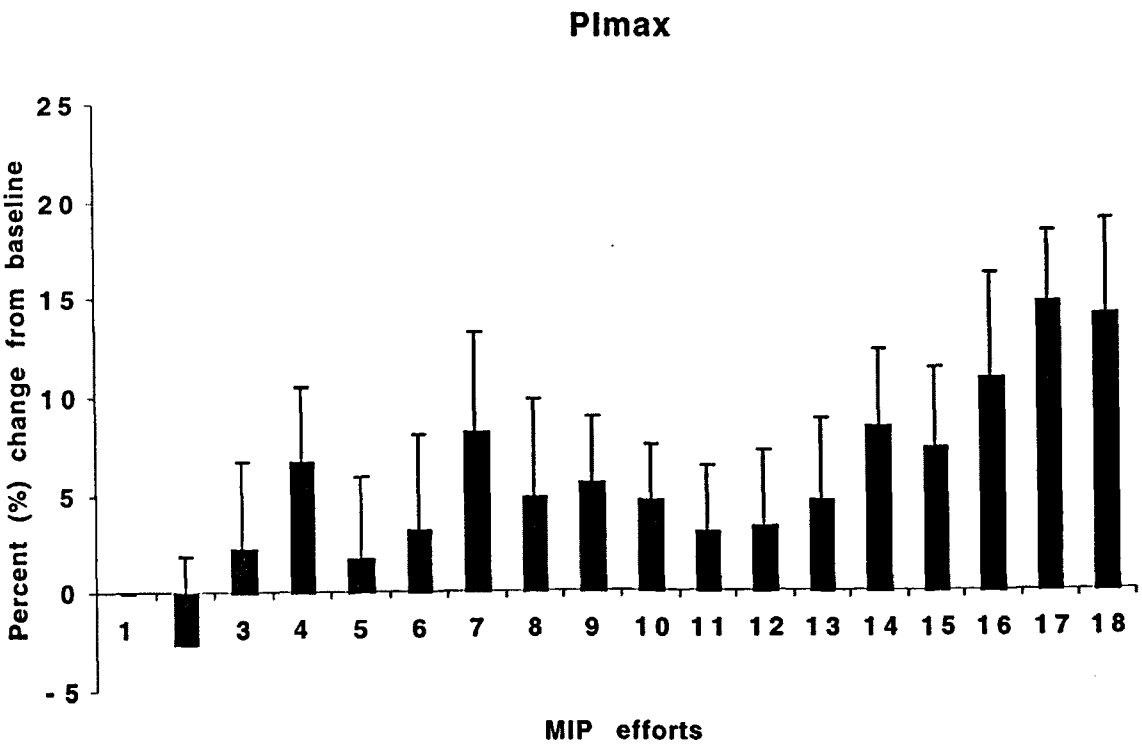
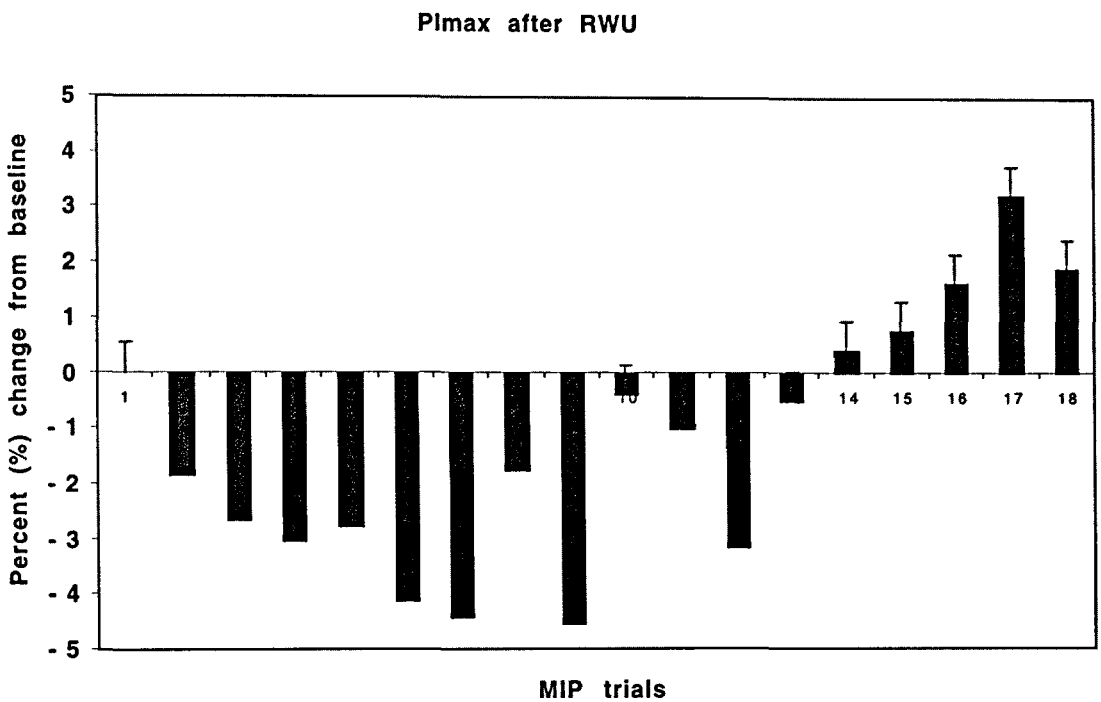


Fig 3.3 Development PImax during the 18 measurement protocol after the RWU.



3-5 DISCUSSION

The main finding of the present study was that following a specific RWU the response to repeated measurement of both PImax and P.PImax was attenuated such that there was no statistical difference between the post-RWU 'baseline' measurement and the 'long' series measurement.

This finding confirmed our original hypothesis and has important implications for the administration of the Mueller manoeuvre and the measurement of inspiratory muscle function. In the control condition, both PImax and P.PImax continued to increase throughout the 18 measurements with significant differences in all stages (Table 3.3). These data are in agreement with previous reports on the 'learning effect' of PImax (Fiz *et al,* 1989; Wen *et al,* 1997) but in contrast with reports of no effect for P.PImax (Wijkstra *et al,* 1995). When the 'baseline' measurement was preceded by a RWU, PImax and P.PImax showed only modest increases of the order of 2-4 cm H_2O across the 18 measurements. This difference was not significant statistically nor, it could be argued, is it significant functionally.

Our data show that following preliminary submaximal activity of the inspiratory muscles (RWU) a reliable baseline value can be measured, for both PImax and P.PImax, with the first trial. However, it is acknowledged that all of our subjects were well acquainted with the Mueller manoeuvre before the 18 trial protocol was initiated. It is possible that, for subjects that are more naive, the RWU may be insufficient to fully enhance PImax. However, the mean increase in PImax over the 18 measurements was around 12% in our subjects, which is comparable with that observed by others in more naive subjects over a similar number of trials (Fiz *et al,* 1989; Wen *et al,* 1997).

The within-session reproducibility of the two conditions was assessed using the coefficient of variation. The coefficients of variation for PImax were similar or

smaller than those reported previously (Aldrich & Spiro, 1995; Maillard *et al,* 1998), whilst those for P.PImax were considerably smaller than the 11.2 % reported by Wijkstra *et al* (1995). The coefficient of repeatability for PImax for the maximum value ('long') was similar to that reported previously (Maillard *et al,* 1998), 25 cm H_2O compared to 28 cm H_2O. The coefficient of repeatability for the 'baseline' measurement was larger than Maillards' (1998), but this can be attributed in part to the differing absolute values of PImax of our subject populations. Our group had mean PImax of 138 cm H_2O whilst Maillards' was 115 cm H_2O. Since the coefficient of repeatability is expressed in absolute values a larger absolute biological variability will be expressed in its values.

It is difficult to identify the mechanism(s) responsible for the increase in PImax and P.PImax following the RWU, but it is unlikely that it was due to the learning effect reported previously (Larson *et al,* 1993). It is possible that part of the observed increase was due to a placebo effect. Whilst the Mueller manoeuvre is a highly effort-dependent test, earlier studies have shown that in well motivated healthy subjects full activation of the diaphragm is possible (Bellemare & Bigland-Ritchie, 1984; Gandevia & McKenzie, 1985). However, a recent study by McKenzie et al (1996) found that voluntary activation of the diaphragm declines during maximal Mueller efforts at volumes below FRC. They conclude that amongst a number of mechanisms which could contribute to the influence of lung volume on voluntary drive to the diaphragm, reflexes dependent on muscle afferents might be involved.

In common with other skeletal muscle, the development of maximal force by the inspiratory muscles requires substantial reflex facilitation (most likely from muscle spindle afferents) in addition to the descending drive (Gandevia *et al,* 1990). It has been suggested that the sudden loading of the inspiratory muscles experienced during the Mueller manoeuvre may produce a reflex inhibition of motorneurones (Butler *et al,* 1995). It is possible that the preliminary respiratory activity of the RWU improves the intramuscular co-ordination and removes some of the reflex

inhibition, resulting in greater force generation. Alternatively, even though the two conditions are different in terms of muscle contraction pattern, the muscle length specificity between the RWU and the Mueller manoeuvre may have contributed to the changed inspiratory muscle performance following RWU. In both test conditions, due care was taken to ensure that the manoeuvres were initiated from RV. This volume represents a specific muscle length of the inspiratory muscles that is not normally involved during quiet breathing. It is quite possible that when a particular movement is repeated many times, alterations occur in the complex interactions amongst muscles, with the result that performance is enhanced (Komi, 1992). Further work is required in this area and the response of trans-diaphragmatic pressure to bi-lateral phrenic nerve stimulation with and without RWU would be of particular interest.

Whilst there were no statistically significant differences between any of the 18 measurements following RWU, there is a suggestion that the RWU may have induced a small degree of fatigue which appeared to recover after about 15 measurements (see figure 3.3). We were not able to calculate the Tension-Time Index (TTI) because the duty cycle (T_I/T_{TOT}) of the breathing pattern was not measured during the RWU. However, as can be seen in the methods, the breathing frequency adopted during the RWU was reduced. The subjects following expiration were pausing at FRC until they felt an urge to breath. This breathing pattern was characterised by a breathing frequency of about 6/min and an estimated T_I/T_{TOT} value of around 0.1. Therefore, we are reasonably confident that the TTI was below the fatigue threshold of 0.15 suggested by Bellemare and Grassino (1982). Even if we accept that some level of fatigue was induced by the RWU the effect that we report is still significant. If the suggested fatigue was prevented the warm-up effect would have been even larger. Therefore we don't believe that the presence, or not, of fatigue is a fundamental limitation of our study or that it should alter our conclusions. In any case, since both protocols resulted in the same absolute inspiratory pressures we can assume that RWU did not induce

any additional fatigue than that, if any, induced by the 18 MIP manoeuvres. Clearly, further work is necessary to identify an optimal RWU which retains the properties of the warm-up utilised in our study.

3-6 CONCLUSIONS

The present study confirms our previous observation that the inspiratory muscles exhibit a 'warm-up' phenomenon following prior submaximal activity. Further, the present data suggest that a specific RWU may negate the so-called 'learning effect' which is one of the main contributors of the test's variability. In both the clinical and academic fields, the use of a RWU may provide a means of obtaining reliable values of PImax and P.PImax following just 3 measurements.

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Chapter Four

Specific Respiratory Warm-Up Improves Rowing Performance and Exertional Dyspnea.

Parts of this chapter have been accepted for publication in *Medicine and Science in Sports and Exercise.*

4-1 ABSTRACT

In a previous study (chapter 2) we have found that the strength of the inspiratory muscles is enhanced following a specific respiratory warm-up. The purpose of this study was a) to compare the effect of three different warm-up protocols upon, rowing performance and perception of dyspnea, b) to investigate the effect of a respiratory warm-up upon rowing performance.

A group of well-trained club rowers (N = 14) performed a 6 min all-out rowing simulation (Concept II). We examined differences in mean power output and dyspnea measures (modified CR-Borg scale) under three different conditions; following a submaximal rowing warm-up (SWU), a specific rowing warm-up (RWU) and a specific rowing warm-up with the addition of a respiratory warm-up (RWUplus) protocol.

Mean power output during the 6 min all-out rowing effort increased by 1.2% following the RWUplus compared to that obtained after the RWU ($P < 0.05$) which, in turn, was by 3.2% higher than the performance after the SWU ($P < 0.01$). Similarly, following the RWUplus, dyspnea was 0.6 ± 0.1($P < 0.05$) units of the Borg scale lower compared to the dyspnea after the RWU and 0.8 ± 0.2 ($P < 0.05$) units lower than the dyspnea following the SWU.

These data suggest that a combination of a respiratory warm-up protocol together with a specific rowing warm-up is more effective than a specific rowing warm-up or a submaximal warm-up alone as a preparation for rowing performance.

4-2 INTRODUCTION

Warm-up may be defined as any preliminary activity that is used to enhance physical performance and to prevent sports-related injuries. There are various types of warm-up techniques that competitors use to prepare for their event. The most widely used methods are classified as *passive, general* and *specific warm-up* (Shellock & Prentice, 1985).

Competitive rowing is considered to be one of the most physiologically demanding sports, as rowers work near their maximal physical capacities and recruit a very large muscle mass. Open class rowers generate amongst the highest values of any athletes in selected physical fitness parameters, including those related to cardiorespiratory and muscular function (Koutedakis & Sharp, 1990). Warm-up is an integral part of the preparation before the start of the race.

Most general warm-up protocols are of moderate intensity and characterised by a low ventilatory demand (Karvonen, 1992). In competitive rowing, however, a higher intensity specific warm-up usually follows the general warm-up in an attempt to practise the racing pace (Grosser, 1991). The higher intensity of the specific warm-up, amongst other peripheral adaptations, elicits an elevated ventilatory response that may prepare the respiratory muscles for the demanding entrained breathing of rowing (Mahler *et al,* 1991; Steinacker *et al,* 1993). However, data from a previous study (chapter 2) have shown that a specific respiratory warm-up protocol is more effective in enhancing inspiratory muscle strength than a whole body specific rowing warm-up protocol (Volianitis *et al,* 1999).

The purpose of this study was a) to compare the effect of three different warm up protocols, b) to identify the functional significance of the respiratory warm-up, in terms of rowing performance and perception of dyspnea.

4-3 METHODS

4-3.1 Subjects

Fourteen competitive club rowers (7 male) participated in the study after giving informed written consent approved by the local Ethics Committee. One of the subjects was removed from the study because he developed a respiratory tract infection within two weeks of the data collection, a condition known to have potential effects on respiratory muscle strength (Mier-Jedrzejowicz *et al,* 1988). Subject characteristics are shown in table 4.1.

Table 4.1. Group characteristics.

(Mean ± SD)	Male (n=7)	Female(n=7)
Age (yr)	19.9 ± 0.7	20.1 ± 0.9
Height (cm)	181.6 ± 5.8	174.7 ± 2.3
Weight (kg)	78.0 ± 10.7	62.9 ± 4.2
FVC (l)	5.7 ± 0.9	4.2 ± 0.3
FEV_1 (l)	4.8 ± 0.8	4.5 ± 0.5
FEV_1/FVC (%)	84.7 ± 7.0	88.3 ± 6.3
$\dot{V}_{O_2}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	61.3 ± 9.0	54.3 ± 2.1

4-3.2 Procedure

Before data collection, all subjects visited the lab on two occasions to be familiarised with mouth pressure, spirometry and dyspnea measurements. The subjects performed three different warm-up protocols, on different occasions, followed by an assessment of rowing performance. The three protocols were a submaximal rowing warm-up (SWU), a specific rowing warm-up (RWU) and the same specific rowing warm-up with the addition of a respiratory warm-up (RWUplus). The respiratory warm-up was performed using a pressure threshold

inspiratory muscle-training device (POWERbreathe®, IMT Technologies Ltd., Birmingham, UK). Rowing performance was assessed with a 6 min all-out effort, on a rowing ergometer (Concept II, model c, Morrisville, USA) as the maximum oxygen uptake ($\dot{V}_{O_2max}$) and average power output obtained from this test are strongly related to competitive rowing (Secher, 1993). Following SWU, the same all-out rowing effort was duplicated on two separate occasions, in order to evaluate the reproducibility of our protocol. Mouth pressure and spirometry measurements where made before and after every protocol. The heart rate was telemetrically monitored with Polar Accurex Plus heart rate monitor (Polar Electro, Finland).

4-3.3 Maximum Inspiratory Pressures (MIP)

MIP is commonly used to measure inspiratory muscle strength. It reflects the force-generating capacity of the combined inspiratory muscles during a brief, quasi-static contraction (Mueller manoeuvre)(Larson *et al,* 1993). MIP was recorded using a portable hand held mouth pressure meter, (Precision Medical, UK). This device has a constant leak to preclude spurious results, due to closure of the glottis and activity of bucal muscles, and has been shown to measure inspiratory efforts accurately and reliably (Hamnegard *et al,* 1994). A minimum of five and a maximum of nine technically satisfactory measurements were conducted and the highest of three measurements with 5% variability or within 5 cm H_2O difference, was defined as maximum (Wen *et al,* 1997). The initial length of the inspiratory muscles was controlled by initiating each effort from residual volume (RV). This procedure was adopted because, from our experience, RV is more reproducible than functional residual capacity (FRC). Subjects were instructed to take their time and to slowly empty their lungs to RV, thereby avoiding problems associated with variability in lung volumes. All manoeuvres were performed in the upright standing position and verbal encouragement was given to assist the subjects to perform maximally. MIP was measured after each

warm-up protocol and following the 6 min all-out effort. During the RWUplus protocol MIP was also measured before and after the specific respiratory warm-up to evaluate its efficacy.

4-3.4 Static Spirometry

Pulmonary function was assessed with a Vitalograph 2120 portable spirometer (Vitalograph Ltd., Buckingham, England), which was calibrated prior to each testing session using a 3 litre calibration syringe (Hans Rudolph Inc., Kansas, USA). Following familiarisation, the best of three manoeuvres were recorded. Forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and percentage expired (i.e. 100 x FEV_1/FVC)(FEV_1 %) were recorded before and after every treatment condition.

4-3.5 Respired Gas Analysis

Breath by breath gas analysis was made with an MGA 2000 Mass Spectrometer (Airspec Ltd., Kent, UK) in conjunction with an ultrasonic phase-shift flowmeter (Birmingham Flowmetrics, Birmingham, UK). Data processing was performed on-line (Labview 3, National Instruments, Austin TX, USA) on a PowerMac 7100/80 computer (Macintosh Ltd., USA). Calibration of the flowmeter was performed before each test using a 1-litre calibration syringe (PK Morgan Ltd., Kent, UK).

4-3.6 Submaximal Warm-Up (SWU)

Eight minutes of submaximal rowing at about 65-70% of the subjects' best previously measured power output during a 6-min all-out effort were performed. The stroke rate was controlled between 22-24 strokes/min. Following the 8-min warm-up there was 3-min rest before the commencement of the 6-min all-out effort. This protocol has been routinely used for physiological assessment of rowers (Two Stage Test)(Steinacker *et al,* 1985). All subjects were familiar with the 6-min all-out effort on the rowing ergometer as part of their training.

4-3.7 Rowing Warm-Up (RWU)

The protocol was designed to mimic as closely as possible the routine that is usually adopted in preparation for a rowing race. Five minutes of very light jogging on the treadmill, at a heart rate of 110-130b/min, were followed by 10 minutes of stretching. Subsequently, 12 minutes rowing of gradually increasing intensity were performed during which the heart rate increased from 148 (± 2) to 178 (± 1.7) b/min. The increase in intensity was achieved primarily by increasing the stroke rate. Then, five sprints with increasing stroke rate and power output were performed. Between each sprint, there was an active rest interval of light paddling which lasted approximately 2 minutes. At the end of the sprints, the rower rested for about 5-7 minutes before any further measurements were made. This rest interval was designed to simulate the small pause between the end of the warm-up and the start of the race. Details of the structure of the Rowing Warm-up can be seen in table 4.2.

Table 4.2. Description of the Rowing Warm-Up on the rowing ergometer.

Warm-up (time)	Stroke rate/min @	Percent Power Max (% Pmax)
1x12min (4-4-3-1)	18-20-22-24	50-55-57-62
2x30s	26-28	77 (± 5) - 80(± 2)
2x45s	28	91 (± 6) - 95(± 2)
1 min	30-32	108 (± 9)

% Pmax = percentage of maximum power output achieved during 6 min all-out test.

This warm-up protocol has been shown to effectively enhance the isokinetic strength of peripheral musculature (Volianitis *et al*, 1999). Breath by breath gas analysis and heart rate data were collected throughout.

4-3.8 Rowing Warm-Up plus respiratory warm-up (RWUplus)

RWUplus was a combined protocol consisting of a rowing warm-up (paragraph 4-3.7) and a specific respiratory warm-up. The specific respiratory warm-up consisted of two sets of 30 breaths using a POWERbreathe® inspiratory muscle trainer (IMT Technologies Ltd., Birmingham, UK) at 40% of the MIP measured before the start of the protocol. Between the two sets there was a short rest interval while an intermediate MIP measurement was made. Forty percent of maximum capacity has been suggested to approximate the upper loading limit before fatigue of the diaphragm occurs (Roussos & Macklem, 1977). POWERbreath® is a pressure-threshold device which requires continuous application of inspiratory pressure throughout inspiration in order for the inspiratory regulating valve to remain open. As with the maximal inspiratory pressures, subjects were instructed to initiate every breath from RV. They continued the inspiratory effort up to the lung volume where the inspiratory capacity for the given resistance limited further excursion of the thorax. Powerful execution of the manoeuvres was encouraged to ensure maximal voluntary output for the given loading conditions. Because of the increased tidal volume, a decreased but spontaneous breathing frequency was adopted by the subjects in order to avoid hyperventilation. This breathing pattern resulted in a very low duty cycle (inspiratory time/total breath duration) and further ensured that fatigue was avoided. The respiratory warm up was performed before the RWU. This protocol has been shown to enhance the strength of the inspiratory muscles (Volianitis *et al,* 1999).

4-3.9 Perception of Dyspnea

A category scale, the modified Borg scale (Borg, 1982), was chosen to evaluate the respiratory effort during exercise. The scale consists of a series of integers from zero to 10. The rower was asked to estimate the effort required to breath but not the effort of the exercise. During rowing, the Borg scale remained in front of the rower and an assessment was made immediately following the all-out effort.

The rowers were asked to assess their dyspnea retrospectively, i.e. during the 6 min effort.

4-3.10 Statistical Analyses

One-way ANOVA with repeated measures and Bonferroni post-hoc test was used to assess differences between the three different warm-up protocols and between the MIP values before and after the different warm-up protocols. Pearson's correlation coefficient was used to assess the association between variables. Values of $P < 0.05$ were considered statistically significant. Data points were means (± SE) unless otherwise stated.

4-4 RESULTS

4-4.1 Test-retest Reliability of the 6min All-Out Effort

Reliability was expressed as a coefficient of variation, (SD/mean) x100, for mean power this was 0.36% and the retest correlation was 0.99 (see table 4.3).

Table 4.3. Coefficients of variation for selected parameters related with the 6 min all-out test.

	CV %
MIP deficit (%)	10.1 ± 2.7
Power (W)	0.36 ± 0.12
Dyspnea	1.7 ± 0.7
$\dot{V}_{O_2}$ (L/min)	1.4 ± 0.7
$\dot{V}_E$ (L/min)	3.2 ± 0.3

4-4.2 MIP Response to the Respiratory Warm-Up

The respiratory warm-up was effective in enhancing the strength of the inspiratory muscles. MIP increased by 7.0 (± 1.0) % from baseline values.

4-4.3 Inspiratory Muscle Fatigue

Following the 6-min all-out rowing effort MIP was lower for all three protocols. After the SWU and the RWU the deficit in inspiratory muscle strength, were 10.2 (± 1.4) and 11.1 (± 1.3)%, respectively. In the RWUplus protocol fatigue was significantly reduced to 4.2 (± 0.3)% ($P < 0.01$) compared with the other two warm-up protocols (see fig.4.1)

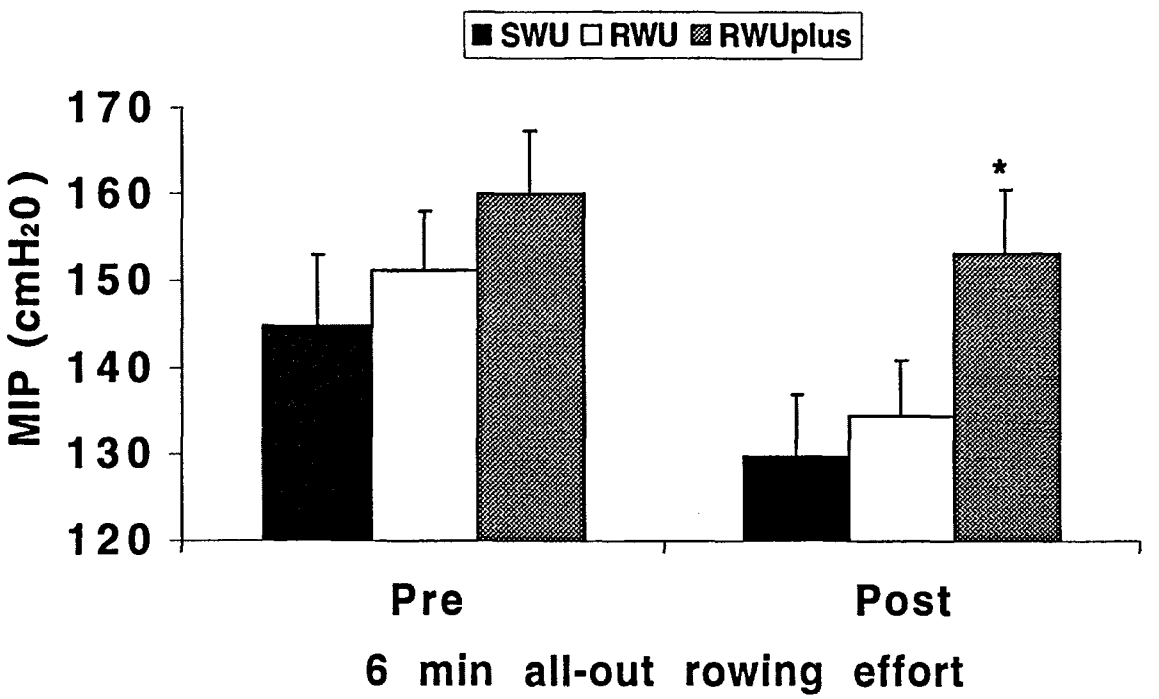

Fig 4.1 Maximum Inspiratory Pressures (MIP) in cmH_2O before and after the 6 min all-out rowing test, for the three different warm-up protocols. Values are means (± SE). * = ($P < 0.05$) Significantly different reduction of baseline MIP from the two other conditions.

4-4.4 Rowing Performance

As can be seen from table 4.4., power output in the 6 min all-out test was 3.2% higher following the RWU compared to power output following the SWU ($P < 0.01$). After the RWUplus, power output increased significantly a further 1.2% compared with the power output following the RWU ($P < 0.05$). The distances covered in meters were increased by 11 (± 15) m ($P< 0.05$) and 18 (± 13) m ($P< 0.01$) following the RWU and RWUplus protocols, respectively, compared with the SWU protocol. There were no significant differences between any gas exchange parameters.

Table 4.4. The effect of the three warm-up protocols on parameters related with the 6min all-out effort.

	SWU	RWU		RWUplus	
MIP deficit (%)	10.2 ± 1.4	11.1 ± 1.3	NS	4.2 ± 0.3	*
Power (W)	292 ±14	302 ± 14	**	305 ± 15	**#
Distance (m)	1690 ± 29	1701 ± 31	*	1708 ± 32	**
$\dot{V}_{O_2}$ (L)	4.17 ± 0.15	4.29 ± 0.21	NS	4.35 ± 0.21	NS
$\dot{V}_E$ (L)	155 ± 5	158 ± 6	NS	160 ± 6	NS
Dyspnea	7.8 ± 0.3	7.6 ± 0.2	NS	7.0 ± 0.3	*

SWU = Submaximal Warm-Up, RWU = Specific Rowing Warm-Up, RWUplus =Specific Rowing Warm-Up with Respiratory warm-up. Values are mean (SE). Comparisons between SWU and RWU. * = P<0.05, ** =P<0.01, # = Significant difference between RWU and RWUplus (P<0.05).

4-4.5 Perception of Dyspnea

The perception of dyspnea during the 6 min all-out effort was not statistically different between the SWU and RWU protocols. However, it was significantly decreased following the RWUplus protocol by 0.8 (± 0.3) and 0.6 (± 0.3) units of the Borg scale compared with the SWU and RWU protocols, respectively ($P < 0.05$). See table 4.4. Even though none of the parameters related to the 6 min all-

out effort were significantly correlated, the association between changes in dyspnea and improvements in power output gave an r = 0.474 which accounts for 22.5% of the variance.

4-5 DISCUSSION

The main finding of this study was that a specific respiratory warm-up has a significant impact upon rowing performance. Indeed, the RWUplus was more effective as a preparatory and warm-up routine for the 6-min all-out effort than both the RWU alone and the SWU protocols.

Reproducibility data for the 6 min all-out effort are in agreement with previous reports suggesting that this test is very reliable and suitable for monitoring rowing performance (Jensen, 1994; Schabort *et al,* 1999). Indeed, high reproducibility was observed in all of the parameters assessed. The coefficient of variation of 0.36% for the average power output, during the 6 min all-out test, is even smaller than the 0.9% reported by Schabort and colleagues.

Respiratory muscle fatigue has been reported following prolonged submaximal exercise (Loke *et al,* 1982), as well as short-term maximal exercise (Johnson *et al,* 1996; McConnell *et al,* 1997). However, it has been suggested that the respiratory muscles of 'athletic' individuals have superior strength and greater fatigue resistance (Coast *et al,* 1990). Nevertheless, the present data suggests that competitive rowers are susceptible to inspiratory muscle fatigue and confirm reports from Johnson and colleagues (Johnson *et al,* 1996) who suggest that a high level of aerobic fitness does not protect the inspiratory muscles from fatigue during heavy exercise. A possible explanation for this respiratory fatigue may be the high ventilatory requirements of rowing. The entrainment of breathing with the stroke rate, observed during rowing, as well as, the dual role assumed by the respiratory, both as actuators of the thoracic expansions and as stabilisers of the thorax for the promotion of external work (Steinacker *et al,* 1993), makes them susceptible to fatigue.

Despite this fatigue, the respiratory muscles as a whole did not reach the point of "task failure" as was evident by the continuous rise of minute ventilation throughout the 6-min all-out test. However, the recruitment pattern of the respiratory muscles might have been altered as a result of this fatigue. Furthermore, the additional motor output to the fatiguing respiratory muscles, necessary to maintain the same pressure generation, would have been perceived as an increased breathing effort and associated dyspnea (Gandevia *et al,* 1981). The respiratory warm-up was effective in enhancing the functional capacity of the inspiratory muscles, confirming our previous findings (Volianitis *et al,* 1999). Following this improved function of the inspiratory muscles, the inspiratory muscle fatigue and the associated dyspnea were decreased. These findings are consistent with previous data (McConnell *et al,* 1997) suggesting that the severity of the inspiratory muscle fatigue is related to their baseline strength. The most likely explanation for this is that greater absolute strength leads to a smaller relative demand for force generation during exercise.

Respiratory sensations are believed to be one subcluster of the overall perceived exertion which is responsible for exercise intolerance (Weiser *et al,* 1973). Moreover, all subclusters are considered interdependent and a significant reduction of the respiratory cluster would improve somewhat, the perceived exertion of the peripheral musculature. A report from Killian *et al* (1992) has shown that, at maximal exercise capacity, dyspnea can be as important, or more so, than leg fatigue in limiting exercise. In this context, the improvements that we have demonstrated in rowing performance during the 6-min all-out test following the RWUplus, may be ascribed, at least partially, to the reductions in dyspnea.

The RWU was more effective as a pre-competitive preparation than the SWU, despite the fact that the intensity and duration of the SWU was sufficient for increasing the body's temperature and inducing the temperature related phenomena of warm-up, as evidenced by the profuse sweating of the subjects.

However, the functional condition of the peripheral musculature is usually neglected in favour of the more centrally oriented adaptations, brought about by temperature increases. Blood flow to the muscles has been shown to increase depending on whether the muscle or muscle fibers (i.e., specificity of muscle fiber type recruited) was used prior to the main exercise (Armstrong, 1988). Therefore, it is possible that the specificity of the RWU in terms of race-pace intensity induced a more pronounced effect of blood flow elevation. Consequently, it could be speculated that both the improved muscle oxygenation and removal of metabolites induced by the increased blood flow, might have contributed the improvements in performance following the RWU protocol.

Another speculation, on the mechanisms responsible for the performance improvements that we observed, might be that the intermittent nature of the RWU was more effective than the equicaloric continuous nature of the SWU in improving the mechanical efficiency and the power output, as suggested by previous reports (Bar-Or, 1987; de Vries, 1994). Therefore, although $\dot{V}_{O_2}$ Peak was not different during the 6 min effort that followed each protocol, improvements in efficiency could have resulted in the observed improvements in power output during the 6-min all-out test after the RWU.

4-6 CONCLUSIONS

In summary, the RWUplus improved the subsequent performance in the 6 min all-out ergometer rowing effort more than the SWU and the RWU protocols. The mechanisms responsible for these improvements are probably associated with the concomitant decreases in dyspnea and inspiratory muscle fatigue. The principle of specificity of adaptive response is exemplified by our findings which suggest that the respiratory muscles should be adequately prepared for optimal performance.

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Chapter Five

Inspiratory Muscle Training Improves Rowing Performance

Parts of this chapter have been accepted for publication in *Medicine and Science in Sports and Exercise.*

5-1 ABSTRACT

We have shown that an acute resistive inspiratory load can enhance the strength of the inspiratory muscles (chapter 2) and subsequently rowing performance (chapter 4). The purpose of this study was to investigate the effects of a long-term inspiratory resistive loading upon rowing performance.

Performance was appraised in fourteen female competitive rowers at the commencement and following 11 weeks of inspiratory muscle training, on a rowing ergometer using a 6min all-out effort and a 5000 m trial IMT consisted of 30 inspiratory efforts twice daily. Each effort required the subject to inspire against a resistance equivalent to 50% peak inspiratory mouth pressure (PImax) using an inspiratory muscle training device. Seven of the rowers, who formed the placebo group, used the same device, but performed 60 breaths once daily with an inspiratory resistance equivalent to 15% PImax.

The inspiratory muscle strength of the training group increased by 44 ± 25 cmH_20 (45.3 ± 29.7 %) compared to only 6 ± 11 cmH_20 (5.3 ± 9.8%) of the placebo group ($P < 0.05$ within and between groups). The distance covered in the 6min all-out effort increased by 3.5 ± 1.2 % in the training group compared with 1.6 ± 1.0 % in the placebo group ($P < 0.05$). The time in the 5000 m trial decreased by 36 ± 9 sec (3.1 ± 0.8 %) in the training group compared with only 11 ± 8 sec (0.9 ± 0.6 %) in the placebo group ($P < 0.05$). Furthermore, the resistance of the training group to inspiratory muscle fatigue following the 6min all-out effort was improved from an 11.2 ± 4.3 % deficit in PImax to only 3.0 ± 1.6 % ($P < 0.05$) pre- and post-intervention, respectively.

IMT and the placebo effect improve rowing performance on the 6min all-out effort and the 5000-m trial.

5-2 INTRODUCTION

Historically, exercise performance has not been considered to be limited by ventilation or respiratory muscle function. However, the occurrence of respiratory muscle fatigue following prolonged submaximal exercise (Loke *et al,* 1982), as well as short-term maximal exercise (Johnson *et al,* 1993; McConnell *et al,* 1997) has suggested that the ventilatory system might contribute to exercise limitation. Some studies in which the inspiratory muscles were partially unloaded during prolonged exercise, and supposedly respiratory muscle fatigue was alleviated, reported no effect on ventilation or exercise performance (Gallagher & Younes, 1989; Krishnan *et al,* 1996), while other studies show significant improvements in performance (Harms *et al,* 1997; Harms *et al,* 1998).

In addition, several studies in recent years have examined the effects of specific respiratory muscle training upon exercise performance but the literature is inconclusive; some have shown improvements (Boutellier *et al,* 1992; Caine & McConnell, 1998; Spengler *et al,* 1999) whilst others show no effect on performance (Hanel & Secher, 1991; Fairbarn *et al,* 1991). The discrepancies between studies may reflect differences in the exercise intensities and durations used for testing, as well as differences in experimental design and fitness level of the subjects.

Rowing is a sport requiring large aerobic power and a high minute ventilation, typically greater than 200 l/min in elite males (McKenzie & Rhodes, 1982). Peak expiratory flow rates can reach values up to 15 l/sec in elite male rowers (Carles *et al,* 1980). The entrainment of breathing in rowing (Steinacker *et al,* 1993) places additional demands on the respiratory muscles, which must stabilize the thorax during the stroke, as well as bringing about breathing related excursions of the thorax. If respiratory muscle fatigue occurs during competitive rowing it might be of physiological significance to the regulation of ventilation and breathing pattern, and

to respiratory muscle recruitment and hence respiratory sensation. Furthermore, an alteration of the recruitment pattern could have an effect on the mechanical efficiencies of breathing and rowing, with detrimental consequences for performance.

In view of the unique respiratory demands of rowing and the discrepancies in the literature with regard to the benefits of inspiratory muscle training, this study investigated the effect of inspiratory muscle training upon rowing performance. The perfomance tests used in this study have a "real world" relevance since they are simulations of rowing competitions and used very frequently by rowers and coaches for training and squad selection.

5-3 METHODS

5-3.1 Subjects

Fourteen female competitive rowers (mean ± SD, age 23.8 ± 3.8 yr, height 173.4 ± 3.8 cm, weight 68.2 ± 4.6 kg, maximal oxygen uptake ($\dot{V}_{O_2 max}$) 3.56 ± 0.17 L/min, maximal power output (Pmax 229 ± 22 W), were assigned randomly to either an inspiratory muscle training (IMT) or placebo group. The subjects were informed about the nature and risks involved in participation in the experiments. The experimental protocol was approved by the local Ethics Committee and all subjects acknowledged voluntary participation through written informed consent. The subjects were instructed to adhere to their usual diet and not to engage in strenuous activity the day before an exercise test. On test days, the subjects were asked not to drink coffee or other caffeine-containing beverages. The tests were performed at similar times of the day. The initial performance assessment took place at the end of October which is the first month of the preparatory period of the rowing season. All the subjects where either national team members or candidates for the national team and had been competing for a minimum of 3-4 years.

5-3.2 Procedure

At the beginning of the study the subjects performed a submaximal incremental load test followed by a 6 minute all-out test on a rowing ergometer (Concept II, model c, Morrisville, USA). On the same occasion, baseline spirometry values and maximum respiratory mouth pressures were taken before and after the rowing tests. Both groups commenced an 11-week period of inspiratory muscle training. The effects of the intervention were evaluated, with the same battery of tests, at 4 weeks and following completion of the training period. Mouth pressure measurements, for evaluation of respiratory muscle function during rowing, took place on all occasions. The manoeuvres were performed within 30 seconds after the completion of the maximum effort.

5-3.3 Submaximal Incremental Load Test

The test protocol consisted of 5 stages of 4 minutes each with a 1min interruption for blood sampling. The initial work rate was individualised based on known work capacity. The rowers where asked to start rowing with a frequency of 18 strokes/min at a work rate that they usually perform their daily warm-up. The work rate increments for each subsequent stage was 20 or 25 W depending on the rower's capacity. Once the protocol for a particular rower was established at the beginning of the study, it was not varied thereafter. Heart rate was monitored via a short-range telemetry system (Polar Sport Tester, Polar Electro, Finland). A pre-exercise and post-stage blood sample was collected from the earlobe and analysed for lactate concentration. Stroke ratings ($st.min^{-1}$), power output (W) were recorded for each stage. Continuous analysis of expired gases and static spirometry (flow-volume loops) were performed with an Oxycon Alpha diagnostic system (Jaeger b.v., Netherlands).

5-3.4 Maximal Performance Tests

Following the submaximal incremental load test, the rowers performed a 6 minute all-out effort which is a simulation of the competitive rowing duration. Rowing events last between 5.5 and 7.5 minutes depending on boat type, category and gender of the rowers. We chose 6 min for our test as it represents the duration of the women's eight event. The rest period between the submaximal test and the 6-minute test was standardised at 8-10 minutes in order to minimize any fatiguing effect of the submaximal test but at the same time to maintain readiness of the rowers. Additional performance data has been obtained at baseline and after 4 weeks of inspiratory muscle training by means of a 5000m ergometer trial which the subjects performed as part of their training control.

5-3.5 Maximum Inspiratory Pressure Measurement

Maximal static inspiratory mouth pressure (PImax) is commonly used to measure inspiratory muscle strength. A portable hand held mouth pressure meter (Precision

Medical, UK) was used for this measurement. This device has been shown to measure inspiratory and expiratory pressures accurately and reliably (Hamnegard *et al,* 1994). A minimum of five technically satisfactory measurements were conducted and the highest of three measurements with less than 5% variability or within 5 cm H_20 (1kPa = 10.3 cm H_20) difference, was defined as maximum (Wen *et al,* 1997). The initial length of the inspiratory muscles was controlled by initiating each effort from residual volume (RV). This procedure was adopted because, from our experience, RV is more reproducible than functional residual capacity (FRC). Subjects were instructed to take their time and to empty their lungs slowly to RV, thereby avoiding problems associated with variability in lung volumes and dynamic airway compression. All manoeuvres were performed in the upright standing position and verbal encouragement was given to help the subjects perform maximally. The subjects had been familiarized with the nature of the manoeuvres in order to reduce any learning effect.

5-3.6 Respiratory Muscle Fatigue

For practical purposes, "fatigue" was defined as the inability to continue to generate a given pressure with the same motor command as when the muscle was still rested. A condition like this does not necessarily imply any "task failure" in the form of inadequate pressure generation for the required ventilation, but it is an indication that the functional capacity is compromised and it will eventually lead to "task failure". Therefore, the original definition of Edwards (1981) of skeletal muscle fatigue as a "failure to maintain the required or expected force" has been extended for respiratory fatigue to include also the state of muscle weakness (National Heart, Lung and Blood Institute, 1990).

5-3.7 Perception of Dyspnea

A category scale, the modified Borg (1982) scale, was chosen to evaluate the respiratory effort during exercise. The scale consisted of a series of integers from 0 to 10. The rower was asked to estimate the effort required to breathe but not the

effort of the exercise. During rowing, the Borg scale remained in front of the rower and an assessment was made at the end of every stage and following the all-out effort.

5-3.8 Inspiratory Muscle Training

The training group performed 30 inspiratory efforts twice daily. Each effort required the subject to inspire against a resistance equivalent to 50% peak inspiratory mouth pressure (PImax) using an inspiratory muscle trainer (POWERbreathe®, IMT Technologies Ltd., Birmingham, UK). POWERbreathe® is a pressure-threshold device which requires continuous application of inspiratory pressure throughout inspiration in order for the inspiratory regulating valve to remain open, while it allows unrestricted expiration. Subjects were instructed to initiate each breath from RV and to continue the inspiratory effort up to the lung volume where the inspiratory muscle force output for the given load limited further excursion of the thorax. Because of the increased tidal volume, a decreased breathing frequency was adopted in order to avoid hyperventilation and the consequent hypocapnia. Previous studies from our lab (Caine & McConnell, 1998b) have suggested that the protocol used by the training group is successful in eliciting an adaptive response. The placebo group trained using the same device, but they performed 60 breaths once daily, at a resistance to inspiration equivalent to 15% PImax, a load known to elicit a negligible training effect (Caine & McConnell, 1998). The two seemingly different training protocols were designed to maintain the naivety of the subjects who were told that one group was training for strength and the other for endurance of the inspiratory muscles. All subjects kept a training diary recording their adherence to the program. Each of the 2 daily sessions of the training group lasted approximately 5 minutes while the single training session of the placebo group lasted approximately 10-12 minutes, depending on the breathing frequency that each subject adopted.

5-3.9 Blood Lactate

Arterialised capillary blood samples were taken from the ear lobe before the incremental load test and at the end of each stage. Analysis was done with an Analox GM7 (London, UK). The within-run precision was 1.6% at a whole blood lactate concentration of 5.0 mmol/l. At low levels of lactate concentration, measurement errors exceeding ± 0.2 mmol/l were rare. Thus, a measured rise of more than 0.4 mmol/l during the course of a progressive test was likely to represent a real increase in lactate concentration.

5-3.10 Statistical Analyses

Results were analysed using non-parametric statistics. Friedman's test and Wilcoxon signed ranks test were used for intra- and inter-group comparisons, respectively. Probability values of less than 0.05 were considered significant. All results are expressed in means ± SD unless otherwise stated.

5-4 RESULTS

5-4.1 Respiratory Muscle Function: PImax

Following the initial 4 weeks of the training period, PImax increased by 40 ± 25 cmH_20 (40.7 ± 25.1 %)($P < 0.01$) and by 5 ± 6 cmH_20 (4.6 ± 6.0 %)($P = 0.083$) from baseline, in the IMT and placebo groups, respectively. After 11 weeks of IMT, PImax increased slightly more to a total increase of 44 ± 25 cmH_20 (45.3 ± 29.7 %)($P < 0.01$) and 6 ± 11 cmH_20 (5.3 ± 9.8%)($P = 0.21$) from baseline, in the IMT and placebo groups, respectively (See table 5.1.) The PImax improvements of the training group, expressed in percentage, were significantly different both between groups and across time within the group. Analysis of the training diaries revealed that both groups compliance with the prescribed training was between 96-97%.

Table 5.1. PImax in centimetres of H_20 (mean ± SE), and performance, in meters (m), during the 6 min all-out rowing effort for the training (IMT) and placebo groups, throughout the 11 weeks of inspiratory muscle training.

	PImax (cm H_20)		Performance (m)	
	IMT	Placebo	IMT	Placebo
Baseline	104 ± 8	130 ± 12	1561 ± 9.3	1566 ± 20.7
4 Weeks	144± 10**	135 ± 11	1613 ± 12.2**	1582 ± 21.4*
11 Weeks	148± 10**	136 ± 12	1616 ± 13.4**	1592 ± 21.1**

Significantly different from baseline ($P < 0.05$),** Significantly different from baseline ($P < 0.01$)

5-4.2 Rowing Performance

6min all-out : After the first 4 weeks of the training period the performance in the 6 min all-out test improved, from baseline, by 3.4 ± 1.0% ($P < 0.05$) in the IMT group, and by 1.1 ± 0.4 % ($P < 0.05$) in the placebo group. The absolute baseline values for the IMT and placebo groups were 228 ± 12 W and 230 ± 25 W respectively. At 4 weeks power in the 6 min all-out test was 242 ± 13 W for the

IMT group and 236 ± 25 for the placebo group. Upon completion of the training period, performance had increased from baseline a total of 3.5 ± 1.2 % ($P < 0.05$) in the IMT group and 1.6 ± 1.0 % ($P < 0.05$) in the placebo group from their baseline values (See table 5.1). Power for the IMT group was 244 ± 22 W and 235 ±26 for the placebo group. These improvements were also significantly different between the two groups after 4 weeks ($P < 0.05$) and after 11 weeks ($P < 0.05$). **5000m:** The time for the completion of the 5000m test, following the first 4 weeks of IMT, decreased by 36 ± 9 sec (3.1 ± 0.8 %)($P < 0.05$) while the placebo group's time decreased by 11 ± 8 sec (0.9 ± 0.6 %)($P < 0.05$). The difference in the improvement between the two groups was also significant ($P < 0.05$). There were no data available for the 5000m test upon completion of the 11 week IMT period.

5-4.3 Lactate

Following 4 weeks of inspiratory muscle training blood lactate was lower relative to baseline values by 0.3 ± 0.3 mmol/L ($P < 0.05$) in the third stage and 1.3 ± 1.3 mmol/L ($P < 0.05$) in the fifth stage of the submaximal incremental test for the IMT group. Even though there was also a decreasing trend in the placebo group it did not reach significance (P = 0.11, in the fifth stage). In the interval between the fourth and eleventh week of inspiratory muscle training blood lactate decreased a further 0.37 ± 0.32 mmol/L ($P < 0.05$) in the IMT group at the second stage of the incremental test with no significant changes in the placebo group. Overall, both IMT and placebo groups had a significant decrease in lactate of 1.3 ± 1.47 mmol/L and 1.3 ± 1.2 mmol/L, respectively ($P < 0.05$) in the fifth stage of the incremental test. There was no significant difference between the groups. No changes occurred in the blood lactate response to the 6 minute all-out effort throughout the study. Absolute blood lactate values can be seen in tables 5.2 and 5.3.

Table 5.2. Blood lactate values (mmol/L) throughout the incremental stage and all-out tests for the three different testing periods of the placebo group.

Period/Stage	1	2	3	4	5	Max
Baseline	0.7±0.3	1.0±0.6	1.7±1.0	3.7±1.5	5.9±1.6	11.2±1.6
4 Weeks	0.6±0.2	0.9±0.3	1.6±0.6	2.9±0.9	4.9±0.9	10.7±2.3
11 Weeks	0.6±0.2	0.8±0.5	1.3±1.0	2.6±1.5	4.6±1.8	10.9±2.1

Table 5.3. Blood lactate values (mmol/L) throughout the incremental stage and all-out test for the three different periods of the IMT group.

Period/Stage	1	2	3	4	5	Max
Baseline	0.8±0.3	0.8±0.4	1.5±0.6	2.4±1.4	4.3±2.0	9.9±2.2
4 Weeks	0.6±0.2	0.7±0.2	1.1±0.6	1.8±1.0	3.0±1.2	9.4±2.0
11 Weeks	0.5±0.3	0.4±0.2	1.1±0.9	2.0±1.7	3.0±2.4	9.5±2.5

5-4.4 Respiratory Muscle Fatigue

Baseline fatigue, defined as the decrease of maximum mouth pressure generating capacity, following the baseline 6 min all-out rowing effort, was 11.2 ± 2.6% ($P <$ 0.05) and 11.1 ± 0.8% ($P < 0.05$) for the IMT and the placebo groups, respectively. After the first 4 weeks of the training period the fatigue following the 6 min all-out effort in the IMT group decreased to 3.1 ± 1.1 % ($P < 0.01$) while the placebo group remained at 10.7 ± 2.8 %. Upon completion of the training period the fatigue for the IMT and the placebo groups did not change any further (4.5 ± 4.7 %, $P <$ 0.01 and 10.7 ± 2.2%, NS, respectively). Between-group differences in fatigue where also significant for both the 4 and 11 weeks comparisons ($P < 0.05$)(See fig. 5.1)

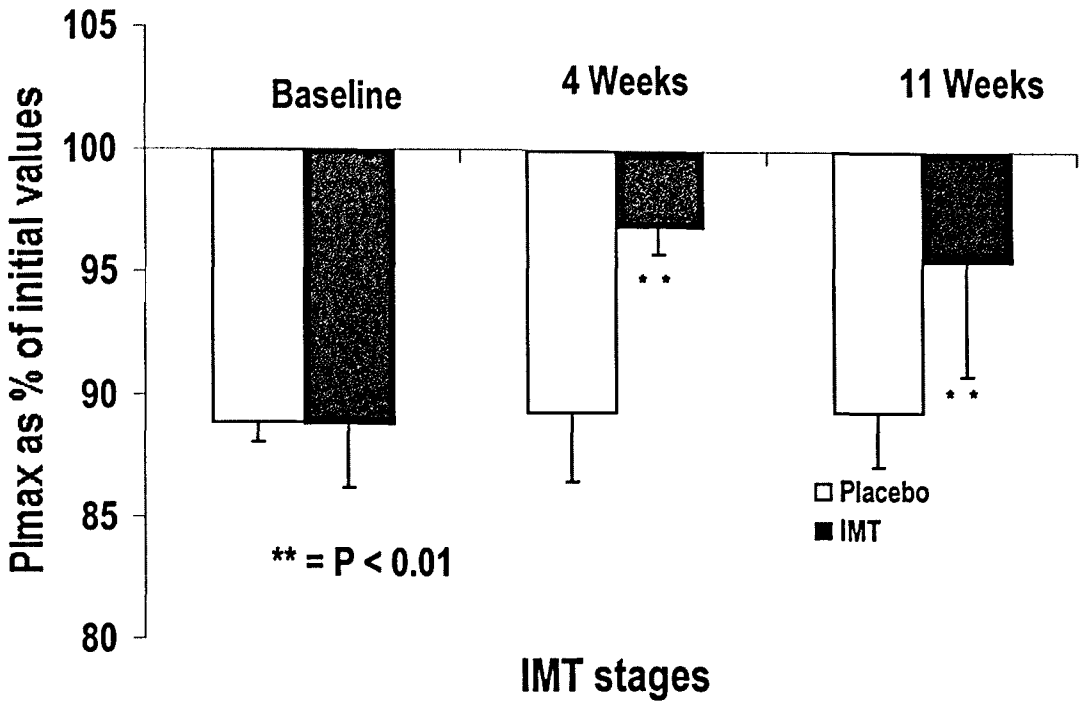

Fig 5.1 Decrement in inspiratory muscle strength following the 6 min all-out test, in percentage decrease from resting mouth pressure generating capacity, throughout the 11 weeks of inspiratory muscle training in the training and placebo groups. Values are mean ± SD. ** $P < 0.01$ different from the placebo group. IMT = Inspiratory Muscle Training group.

5-4.5 Perception of Dyspnea

Significant improvements in the perception of respiratory effort during the incremental test were found in the IMT group throughout the training period (Fig. 5.2). However, no change was found in the dyspnea following the 6 minute all-out effort. There were no significant changes in the control group either during the incremental test or the 6 minute all-out effort (Fig. 5.3).

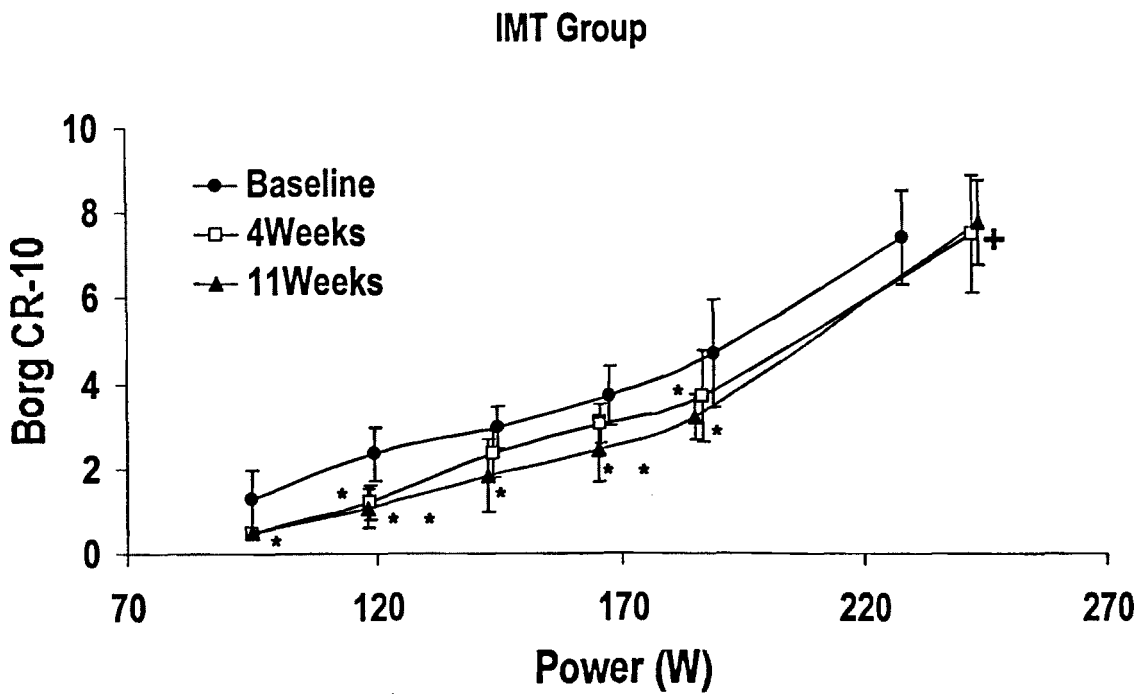

Fig 5.2. Dyspnea-Power curves after 4 and 11 weeks of inspiratory muscle training for the IMT group. Values are means ± SD. * Significantly different ($P < 0.05$), ** significantly different ($P < 0.01$). Note: + significantly different ($P < 0.05$) power output for the same dyspnea.

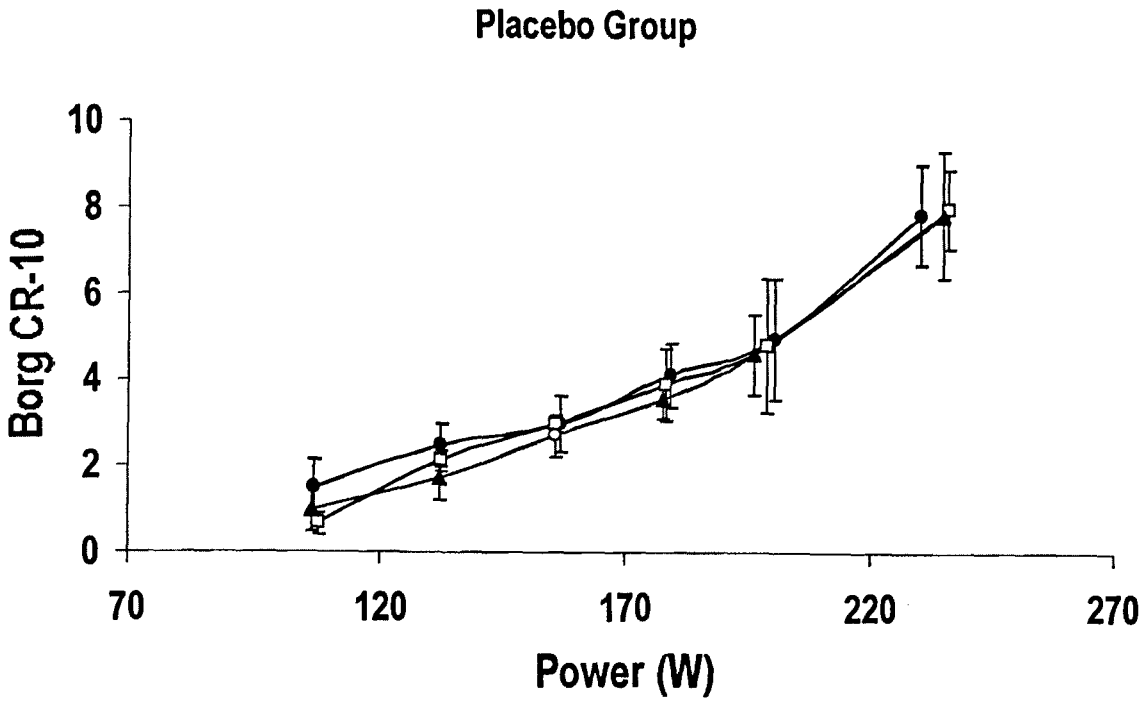

Fig 5.3. Dyspnea-Power curves after 4 and 11 weeks of inspiratory muscle training for the Placebo group. Values are means ± SD. * Significantly different ($P < 0.05$), ** significantly different ($P < 0.01$). Note: + significantly different ($P < 0.05$) power output for the same dyspnea.

5-4.6 Ventilation and Breathing Pattern

Following the completion of the training period, there were no significant changes in the ventilatory volumes at any stage of the incremental test, for either the IMT or the placebo group. However, during the 6min all-out effort, minute ventilation increased for the placebo group, from a baseline of 120.3 ± 18.5 to 129.6 ± 13.4 l/min ($P < 0.05$). The IMT group also increased minute ventilation from a baseline of 119.9 ± 12.8 to 122.5 ± 12.3 l/min, a difference which just failed to reach significance ($P = 0.051$). The breathing pattern of the IMT group at the 6 minute all-out effort changed following the completion of the training period. There was a shift to a significantly deeper breathing pattern with an increase of the tidal volume from 2.01 ± 0.16 to 2.16 ± 0.16 l ($P <0.01$). Breathing frequency did not change significantly. The placebo group did not exhibit any significant changes in breathing pattern, but there was a tendency towards a more tachypneic pattern

with an increase of 4.5 % in their breathing frequency compared to only 1.5 % of the IMT group.

Table 5.2. A summary of statistical significance for within and between-groups comparisons following 11 weeks of IMT in selected parameters.

Parameter	IMT group	Placebo group	Between Group Comparisons
Resting PImax	Improved	No change	Different
PImax after exercise	Improved	No change	Different
Lactate Incremental test	Decreased	Decreased	No difference
Borg scale 6min test	Decreased	No change	No difference
$\dot{V}_{O_2max}$ 6min test	Increased	Increased	No difference
$\dot{V}_E$ Incremental test	No change	No change	No difference
$\dot{V}_E$ 6 min test	No change	Increased	No difference
V_T 6 min test	Increased	No change	No difference
f_R 6 min test	No change	No change	No difference
$P_{ET_{CO_2}}$	No change	No change	No difference
$P_{ET_{O_2}}$	Increased	No change	Different
6 min test Power	Improved	Improved	Different
5000m trial Time	Improved	Improved	Different

5-5 DISCUSSION

The most important finding of this study is that inspiratory muscle training improved rowing performance to a greater extent than conventional training alone. To our knowledge, ours is the only study investigating the effect of inspiratory muscle training upon an index of sports performance rather than a marker of physiological capacity such as the time-limit test (T_{lim}). In the reports of Caine *et al* (1998) and Lisboa *et al* (1997), cycling time to exhaustion, a 6 min walk, or an incremental test are used for evaluation of exercise tolerance. Even though the 6 min walk might be argued to be representative of a task encountered by patients with COPD, it is still not a simulation of any known sport. In contrast, the 6 min and the 5000 m time trial, represent very close simulations of competitive rowing events, and are therefore one step closer to actual sports performance than any test attempted in previous studies.

Since the early report of Leith & Bradley (1976) many different groups have demonstrated that ventilatory muscle training increases maximal voluntary ventilation, ventilatory muscle strength, ventilatory muscle endurance and functional exercise capacity. Our results, of 45.3 % improvement in PImax, are similar in magnitude to other studies, (see Smith *et al* (1992) meta-analysis on patients with COPD) ranging from 32% to 53% (Suzuki *et al*,1993; Lisboa *et al*, 1997; Weiner *et al*, 1998). However, since there may well be important differences between healthy subjects and those with COPD, a more appropriate comparison would be with studies using healthy subjects (Hanel & Secher, 1991; Caine & McConnell, 1998) where PImax also increases in the range of 34% to 45.3%, respectively following 4 weeks of inspiratory muscle training.

Previous reports (Boutellier *et al*, 1992; Caine & McConnell, 1998; Spengler *et al*, 1999) have shown that following inspiratory muscle training a submaximal power

output can be maintained for longer (T_{lim} test). However, the intensity used for the T_{lim} test in these studies was associated either with the anaerobic threshold (Th_{an}) or the maximum lactate steady state (MLSS). Even though these physiological markers correlate very well with endurance performance, this approach is one step removed from competitive sports performance. Our study shows that inspiratory muscle training can improve performance in 2 tests that simulate competitive performance as closely as possible in the laboratory context, viz. the 6 min all-out effort and the 5000m trial. Both tests are routinely used for rowing-specific performance evaluation by coaches. Both IMT and placebo groups improved their performance following 11 weeks of training. The margin of their improvement was expected because the study commenced at the beginning of the preparatory training period and lasted for the bigger part of it. Even though we acknowledge the possibility that the responses observed may have occurred as a result of the subjects' regular training, the 1.9% improvement of the IMT group in the 6 min all-out effort over and above the improvement of the placebo group suggests that this is unlikely. Therefore, the data suggest that the inspiratory muscle training had an additional effect upon rowing performance beyond that expected by regular training. The significance of this difference can be appreciated more within the context of competitive rowing where Olympic medals are decided with a much smaller margin than 1.9%.

We believe that there are a number of reasons why other studies have not reported any significant improvements in performance following IMT. Arguably, the most important of which is the low reliability of the tests used to evaluate performance in other studies, compared with the 6min all-out effort used in our study, made the detection of a meaningful effect difficult. For example, the coefficient of variation for the T_{lim} test has been reported to be anything between 25-40%, while the 6 min all out test is only 2.4% (Jensen, 1994). Therefore, much larger improvements were required to assure that the observations were not due to the variability of the test itself. Other studies (Hanel & Secher, 1991; Fairbarn *et*

al, 1991) have reported improvements in performance but failed to reach significance. We suspect that insufficient statistical power, due to the small sample size of these studies, may have introduced a type II error and failed to reject the null hypothesis. Support of our findings is provided by studies using isocapnic hyperpnea training protocols which suggest that respiratory muscle training induces significant improvements in cycling performance (T_{lim}) (Boutellier *et al,* 1992; Spengler *et al,* 1999). In addition, a recently completed study showed that following 5 weeks of respiratory muscle training, using a high velocity (flow) and a high resistance (pressure) training protocol, cycling time trials improved significantly by approximately 2% (J. Dempsey, personal communication).

In the absence of any clear insight into the hard evidence of the underlying physiological mechanisms for the observed effects, we are forced to speculate on possible mechanisms, three of which are discussed below.

"*Respiratory Muscle Fatigue*". First, even though respiratory muscle fatigue of the IMT group was diminished there was no evidence for significantly different ventilatory response between the two groups. These data support the notion that respiratory muscle fatigue was without significant consequence for the ventilatory response. This is consistent with the suggestion that when the diaphragm is confronted by fatiguing contraction patterns, the accessory inspiratory muscles become more active and the overall ventilation is not compromised. Therefore, since the respiratory pump did not fatigue to the point of "task failure", it is unlikely that the improvements in performance were the result of improved gas exchange or a better compensation for metabolic acidosis. However, the altered breathing pattern observed following IMT suggests that respiratory muscle fatigue might have been of physiological significance to the regulation of the breathing pattern. In the IMT group tidal volume increased significantly, whilst the placebo group resorted to a more tachypneic breathing pattern, characteristic of respiratory muscle fatigue for the maintenance of minute ventilation. Indeed, as the breathing

pattern during exercise seems to be optimised in order to avoid exhaustive fatigue and "task failure" of the respiratory muscles, the increased strength of the IMT group might have enabled them to increase tidal volume without fatiguing. In contrast, the placebo group, which was susceptible to fatigue, resorted to an increased breathing frequency. Even though we did not assess the degree of entrainment between breathing and stroke rate, it is possible that the prevention of a tachypneic breathing pattern in the IMT group enhanced the mechanical efficiency of the rowing work by enabling the maintenance of entrainment. Indeed, our data are in agreement with previous suggestions that breathing in rowing occurs at times where muscle synergy produces larger ventilatory volumes for a given amount of respiratory work, or alternatively, the same volume for less respiratory work (Siegmund *et al,* 1999); consequently performance may be improved.

"Altered Respiratory Sensation". The second putative mechanism for the improved rowing performance may be that the reduced respiratory muscle fatigue induced changes in the respiratory sensation. Respiratory muscle fatigue has been documented following prolonged submaximal exercise (Loke *et al,* 1982) as well as short-term maximal exercise (Johnson *et al,* 1993; McConnell *et al,* 1997). There is some suggestion that the respiratory muscles of 'athletic' individuals have superior strength and greater fatigue resistance (Coast *et al,* 1990). Our data showing significant inspiratory muscle fatigue following a 6 min all-out rowing effort is in agreement with Johnson and colleagues (Johnson *et al,* 1996) who suggest that a high level of fitness does not protect the diaphragm muscle from fatigue during heavy exercise (95 % of $\dot{V}_{O_2max}$). Following inspiratory muscle training the IMT group showed significantly reduced fatigue after the 6 min all-out effort. Indeed, a recent report has shown that the baseline strength of the inspiratory muscles influences their fatigueability (McConnell *et al,* 1997). Interestingly, the fatigue of the placebo group remained the same which suggests that normal

training for rowing does not elicit the same adaptations as a specific inspiratory muscle training program. The increase in strength may have attenuated the development of fatigue by decreasing the proportion of the maximal force capacity required for each breath (Hickson *et al,* 1988). Similarly, with greater inspiratory muscle strength, a smaller fraction of maximum tension is generated with each breath and it has been suggested that this reduces the motor output to the respiratory muscles and decreases the perceived sense of respiratory effort (El-Manshawi *et al,* 1986). Even though we do not have measures of dyspnea during the 5000m test, when asked to describe their feeling afterwards most subjects said that either the onset of breathlessness was delayed, allowing a longer maintenance of the previous pace, or a higher pace was kept throughout the test with the same breathing effort.

"Altered Ventilatory Efficiency". Finally, it has been suggested that through inspiratory muscle training an increase in the mechanical efficiency of ventilation might take place, thereby reducing the metabolic requirements of the respiratory muscles. Previous studies have shown that during maximal exercise the $\dot{V}_{O_2}$ of the respiratory pump can reach values up to 15% of the total $\dot{V}_{O_2}$ (Aaron *et al,* 1992; Aaron *et al,* 1992b). Indeed, the metabolic cost of breathing becomes so great that any additional increase in total $\dot{V}_{O_2}$ contributes minimally to the external work. In studies conducted at $\dot{V}_{O_2 max}$, the respiratory muscles have been perceived as "stealing" blood flow from the peripheral musculature to cover their metabolic requirements (Harms *et al,* 1997). Thus, decreasing the metabolic requirements of the inspiratory muscles could result in a diminished blood flow demand and reduce the competition with the locomotor muscles for limited blood flow. Since we did not see any significant differences in the $\dot{V}_{O_2 max}$; by implication cardiac output was also unchanged. Thus, we can assume that the fraction of the total cardiac output

distributed to leg muscles may have increased following IMT and this may have led to improvements in performance (Harms *et al,* 1998).

5-6 CONCLUSIONS

In summary, significant improvements in the 6min all-out effort and 5000m time trial performance were observed following a period of inspiratory muscle training. These performance improvements were accompanied by a decrease in inspiratory muscle fatigue and perception of dyspnea. Even though the small sample size does not allow us to make inferences about the population from which the sample was drawn, it has not escaped our attention that our findings may have some bearing on rowing performance. The elucidation of the precise mechanisms responsible for our observations requires further studies involving the cardiovascular consequences of inspiratory muscle training and larger sample sizes.

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Chapter Six

General Discussion

6.1 Summary

In this section we will first summarise the major findings of this thesis and discuss on the possible mechanisms responsible for them.

The main focus of this thesis was to test the hypothesis that, "chronic and acute loading of the inspiratory muscles of competitive rowers bestows improvements in inspiratory muscle strength and rowing performance". To this end a series of research findings have been presented. These include:

1. Assessment of the reproducibility of the 6 min all-out rowing effort and development of a testing protocol to maximise the reliability of maximum inspiratory mouth pressure measurement.
2. Evaluation of the effectiveness of different warm-up protocols on inspiratory muscle strength.
3. Evaluation of the effect of a specific respiratory warm-up upon rowing performance, perception of dyspnea and comparison of two different whole body warm-up protocols
4. Evaluation of the effect of 11 weeks of inspiratory muscle training upon rowing performance and the perception of dyspnea.
5. Investigation of the incidence of inspiratory muscle fatigue following a 6 min all-out rowing effort.

6-2 Assessment of the Reproducibility of the 6 min All-Out Rowing Effort

Very high reproducibility was demonstrated for the 6 min all-out effort. A determining factor for the high reproducibility may have been the high external validity of this test as a simulation of competitive rowing. The subjects used were all experienced rowers and they were all very familiar with the nature of the test and the pacing strategy required to produce a maximum effort within the 6min. The fact that the 6min all-out effort has been used regularly by the subjects for

their training may also explain the high reproducibility observed. Another possible factor for the high reproducibility is the inherent stability of the rowing ergometer. The fixed resistance used to produce the workload and the absence of any requirement for calibration may influence the reproducibility of the ergometer. Whatever the reason of the high reproducibility, the 6 min all-out effort has been shown to be suitable for monitoring rowing performance.

6-3 Evaluation of the Effectiveness of Different Warm-Up Protocols on Inspiratory Muscle Strength

The main finding was that inspiratory muscle strength can be more effectively optimised using a specific respiratory warm-up than two differing whole body warm-up protocols. This is a novel concept that shows that the respiratory muscles are capable of the same acute responses as other skeletal muscles following preliminary activity.

The two whole body warm-up protocols included both a moderate and a higher exercise intensity. However, the ventilatory response of both protocols failed to impose a stimulus sufficient to improve muscular strength. In contrast, the use of an inspiratory threshold load improved inspiratory muscle strength significantly. It is possible that the required stimulus for the observed inspiratory muscle warm-up effect, may be related to the volume of respiratory work in the form of hyperventilation or inspiratory resistive loading (i.e., elevated respiratory motor output). However, since the hyperventilatory response occurs only at high and sustained rowing intensity, it appears that both of the whole body warm-up protocols were inadequate in this respect. In fact, most warm-up routines aim to optimise performance components with minimal induced fatigue, thus avoiding prolonged high intensity rowing and the associated hyperventilatory response.

A proposed mechanism for the observation that a specific respiratory warm optimised the global inspiratory pressure generating capacity is that the specificity of the respiratory warm-up improved the neuromuscular co-ordination utilised by the complex respiratory "pump".

6-4 Development of a Testing Protocol to Maximise the Reliability of Maximum Inspiratory Mouth Pressure Measurement.

We found that a specific respiratory warm attenuates the learning effect observed with repeated Mueller manoeuvres and a more reliable baseline is established. Previous studies which have failed to show an exercise-induced respiratory muscle fatigue by measuring maximum mouth pressures may have been deluded by not establishing a reliable baseline (Warren *et al,* 1989; Hill *et al,* 1991). Our protocol may be more important in raising a methodological issue in respiratory studies than changing the clinical practice but, nevertheless clinicians should be aware of the possibility that respiratory fatigue may be "masked".

6-5 Evaluation of the Effect of a Specific Respiratory Warm-Up Upon Rowing Performance and Comparison of Two Different Whole Body Warm-Up Protocols

The main finding of this study was that a specific respiratory warm-up has a significant impact upon rowing performance. Indeed, the protocol that combined a specific rowing warm-up and a specific respiratory warm-up was more effective as a preparatory warm-up routine for the 6-min all-out rowing effort than either the specific rowing warm-up alone, or the submaximal warm-up protocol. In addition, the specific rowing warm-up was more effective in enhancing the subsequent rowing performance than the submaximal rowing warm-up.

For an appreciation of the suggested mechanisms by which the different warm-up protocols may affect rowing performance it is appropriate to examine some of the potentially limiting factors of rowing performance:

6-5.1 Subjective Symptoms of Fatigue as a Limiting Factor in Rowing

Among the most pronounced symptoms of exercise intolerance is the sensation of fatigue. Subjective correlates of fatigue are complex, reflecting the integration of many discrete sensations having different physiological origins. A model of fatigue has been identified consisting of three relatively unique subsets, namely leg fatigue, general fatigue, and cardiopulmonary fatigue (i.e., sensation of dyspnea) (Weiser *et al,* 1973). The leg fatigue subcluster is presumed to be task specific to submaximal cycling to exhaustion which was used as the exercise mode for the construction of the model. While there is an absence of similar studies using rowing as the exercise mode, it is reasonable to assume that peripheral muscle fatigue is also a determinant subcluster in the overall sensation of fatigue in rowing. Furthermore, Horstman *et al,* (1979) concluded that these subjective symptoms arising form muscles, joints, and the cardiorespiratory system operate in consort with physiological processes to set the upper limits of endurance performance.

Sensations of fatigue are equally important as physiological determinants of fatigue in setting the limits of exercise performance (Kinsman & Weiser, 1976). The decision to continue or discontinue exercise is based on subjective symptoms that have their origin in underlying physiological events. These physiological events involve both the contractile functions of peripheral skeletal muscle and cardiorespiratory responses. During exercise functional changes in the neuromuscular and cardiorespiratory systems increase symptom severity, thereby subjectively setting the limits of endurance performance. This assumption has

been validated for cycling (Weiser & Stamper, 1977) but no data exists for rowing performance.

Data from our studies suggest that the perception of dyspnea may be an important determinant of rowing performance. Indeed, alleviation of dyspnea subsequent to a specific respiratory warm-up was associated with improved rowing performance.

6-5.2 Peripheral Mediators of Exertion

Peripheral physiological mediators are primarily regionalised to exercising muscles in the limbs, trunk, and upper torso (including the shoulder and neck). The processes thought to mediate the intensity of peripheral exertional perceptions are 1) metabolic acidosis (pH and lactate), 2) fast- and slow-twitch contractile properties of skeletal muscle fibre, 3) muscle blood flow, and 4) blood-bourne energy substrates (Noble & Robertson, 1996). It is beyond the scope of this thesis to analyse all these factors but for our purposes it is important to emphasise that a reduction in blood flow exacerbates peripheral muscle fatigue and intensifies peripheral exertional signals. Indeed, findings from Harms *et al* (2000) suggest that the reduction in leg blood flow, associated with the work of breathing normally incurred during sustained heavy-intensity exercise (> 90% $\dot{V}_{O_2\,max}$) enhances both the onset of leg fatigue and the intensity with which both leg and respiratory muscle efforts are perceived. The combination of these factors has a significant influence on exercise performance.

6-5.3 Possible Mechanism for the Effect of Warm-Up on Rowing Performance

It is possible that a specific rowing warm-up characterised by a specific muscle fibre recruitment pattern enhances (more effectively than other protocols) the blood flow and speeds $\dot{V}_{O_2}$ kinetics in the active musculature. In turn, the increased

blood flow not only enhances muscle function but also reduces perceived exertion. Additionally, the specific warm up is a rehearsal of the physical, technical, and tactical performance requirements. In this way, attentional focus and arousal levels are optimised.

Altered Central Perception

Alternatively or additionally, an altered central perception of the breathing discomfort, i.e., desensitization to dyspnea may take place following the inspiratory muscle warm-up. Indeed, a study by Revelette & Wiley (1987) investigated the long-lasting plasticity of respiratory sensation. They found that after a 2 min period in which subjects were required to breathe through a high resistive load, the perceptual scores for a loading protocol were reduced compared with those before high resistance breathing. A later study by Wilson & Jones (1990) found that subjects could be conditioned to decrease the perceived magnitude of their dyspnea in an exercise test by the presence of a small inspiratory resistance prior to the test.

In conclusion, desensitisation of respiratory sensations may be responsible not only for a decrement in perceived dyspnea but also in the perceived peripheral effort. These responses induce a general state of readiness which is an important prerequisite for conscious, active and goal-oriented activity and ultimately, may improve rowing performance.

6-6 Evaluation of the Effect of 11 Weeks of Inspiratory Muscle Training Upon Rowing Performance and the Perception of Dyspnea

The main finding of this study was that inspiratory muscle training can have a significant effect upon rowing performance. Performance in both the 6min all-out effort and the 5000m trial improved following 11 weeks of inspiratory muscle

training. The distance covered in the 6min all-out effort increased by 3.5 ± 1.2 % in the training group compared with 1.6 ± 1.0 % in the placebo group. The time in the 5000 m trial decreased by 36 ± 9 sec (3.1 ± 0.8 %) in the training group compared with only 11 ± 8 sec (0.9 ± 0.6 %) in the placebo group. In addition, perception of dyspnea was reduced in both the incremental test and the 6min all-out effort.

While definitive data demonstrating IMT induced hypertrophy in healthy humans do not exist, numerous studies, including those presented within this thesis, have demonstrated that IMT increases the peak force generating capacity of the inspiratory muscles. In addition, McCool *et al* (1997) concluded that the diaphragmatic cross-sectional area in healthy subjects can be increased by long term general or specific training. Given that specific IMT regimes have consistently augmented maximum inspiratory pressure (MIP) values it is reasonable to assume that the respiratory muscles respond in a similar manner to a progressive overloading stimulus to other skeletal muscle; that being to hypertrophy over a period of time.

With a higher absolute MIP capacity a smaller percentage of this capacity will be used to meet a given pressure requirement related with the hyperpneic response to rowing. Since the incidence of respiratory muscle fatigue has been related to the ratio of produced pressure to pressure generating capacity, fatigue of the inspiratory muscles should be decreased, if not prevented completely after training. Consequently, the increase in respiratory motor command outflow, secondary to the degenerating capacity of the respiratory muscles, should be avoided. As a result the perception of dyspnea should be relatively low and the overall perception of exertion improved. This hypothesis is in agreement with our findings.

6-6.1 Improved Breathing Pattern

The hyperventilatory response of high intensity exercise is characterised by an altered respiratory muscle recruitment (Aliverti *et al*, 1997). The diaphragmatic pressure production plateaus while the ventilatory pressure requirements continue to rise, presumably supported by increased accessory muscle activity. The altered recruitment model leads progressively to a tachypneic breathing pattern characterised by a curtailment of tidal volume and increased frequency. This pattern has been interpreted as a sign of respiratory fatigue. The mechanisms underlying this alteration in breathing pattern remain speculative, This tachypneic pattern is mechanically and metabolically inefficient and results in exponential rises of the total work of breathing and consequently of the blood flow requirements of the respiratory muscles. It can be hypothesised that a less tachypneic breathing pattern would uphold the metabolic cost of the respiratory muscles and favourably affect exercise performance.

After IMT, the training group was able to increase minute ventilation through further increases in tidal volume, whilst the control group had to resort to a tachypneic breathing pattern. Thus, improved functional capacity of the inspiratory muscles was manifested through a maintenance of a more efficient breathing pattern throughout the 6 min all-out effort. The benefits of such an adaptation may be twofold.

First, if we accept that the purpose of the entrained breathing pattern in rowing is to support the production of the external work, rowing efficiency may be optimised or at least maintained at the initial levels throughout the test. Secondly, a more efficient breathing pattern may have reduced energy requirements of the respiratory muscles and "spared" a portion of the cardiac output, otherwise allocated to them, for use by peripheral musculature.

6-7 Investigation of the Incidence of Inspiratory Muscle Fatigue Following the 6 min All-Out Rowing Effort

Significant reductions of maximum inspiratory pressure were observed following the 6 min all-out rowing effort. MIP decreased from 142.8 (± 11.2) cmH_2O baseline value to 126.8 (± 37.2) cmH_2O post exercise, corresponding to a decrease of 11.2 ± 4.3 %.

Although the measurement of maximum mouth pressure is a volitional index of the respiratory muscle generating capacity, it is a very useful non-invasive technique which, if performed by fully motivated subjects, can provide a profile of the global respiratory muscle capacity. We are reluctant to conclude that these pressure reductions represent fatigue of the diaphragm but nevertheless, we can conclude that the overall volitional mouth pressure generating capacity was reduced. The reduction of the volitional capacity is more functionally relevant for the performance of the global respiratory system since the isolated fatigue of the diaphragm does not necessarily imply failure of the global respiratory "pump".

6-8 Future studies

The question of dose-response in inspiratory muscle training should be addressed. Future studies should evaluate the effect of different training protocols on the function of the respiratory muscles and rowing performance. In addition, the detraining effect should be investigated to establish the degree to which the adaptations are maintained. The question of diaphragmatic fatigue in rowing with the use of phrenic nerve stimulation following maximal rowing efforts should also be addressed in future investigations.

Studies of a more invasive nature could investigate the mechanisms responsible for the improvements in performance. Investigations of the cardiovascular

consequences of inspiratory muscle training and specifically measurements of leg blood flow following inspiratory muscle training may give us some definitive answers to the suggested mechanism of blood flow redistribution.

Future studies should investigate the effect of sympathoexcitatory reflexes from the diaphragm and accessory respiratory muscles, activated under fatiguing conditions, upon the regulation of limb vascular resistance and blood flow in limb locomotor muscles during exercise. It is hoped that these studies will provide much needed insights into the poorly understood, complex relationships between respiration and circulation during exercise that we believe are critical to controlling blood flow distribution, cardiac output and exercise performance in healthy humans and in those with heart failure and respiratory disease.

6-9 References

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Appendix

Structure and Function of the Respiratory Muscles

A.1 Anatomy

The diaphragm , the external and internal intercostals and the principal accessory (abdominal, scalenes, sternocleidomastoid) muscles of respiration are morphologically, and functionally, the same as all skeletal muscles. The only distinguishing features are possibly their anatomy and patterns of use (Jones, 1995). The diaphragm is internal to the ribs at its attachments and continuous with the innermost muscle layer of the abdominal wall. The thoracic wall muscles, in the plane of the ribs, are represented by the internal and external intercostals. The muscle layers external to the ribs stabilise the pectoral girdle and arm, and almost completely cloak the external surface of the thoracic cage.

A.1.1 The Diaphragm

The diaphragm is a thin musculotendinous sheet of complex structure which forms the floor of the thoracic cavity. It is attached peripherally around the complete boundary of the inferior aperture, from which it passes upward, ascending high into the thoracic cavity to its two cupolae. The muscle of the diaphragm, all of which inserts into a roughly trilobed central tendon, falls into two main components, costal and crural according to its site of origin.

Functionally, the diaphragm can be considered as an elliptical cylindroid, capped by a dome. The dome of the diaphragm corresponds primarily to the central tendon, and the cylindrical portion corresponds to the muscle directly opposed to the inner aspect of the lower rib cage, the region termed the zone of apposition (Mead, 1979). When tension increases within the diaphragmatic muscle fibers, a caudally oriented force is applied on the central tendon, and the dome of the diaphragm descends. This has two effects, firstly, it expands the thoracic cavity along its craniocaudal axis, secondly it produces a caudal displacement of the abdominal visceral mass.

A.1.2 The Intercostal Muscles

There are 11 pairs of internal and external intercostals which fill the space between the ribs. The external intercostal run obliquely inferiorly and anteriorly beginning lateral to the rib tubercules and extending anteriorly to their costal cartilages. The internal intercostals run at right angles to the external intercostals with their fibers run inferiorly and posteriorly. When an intercostal muscle contracts it pulls the upper rib down and the lower rib up. However, the distinct orientations of the muscle fibers coupled with the different insertion points alter the torque characteristics upon each respective rib. Therefore, the external intercostals exert a greater force on the lower rib and produce an inspiratory action since they help raise the inferiorly positioned rib. On the other hand, the internal intercostals produce a downward movement of the ribs and therefore, considered as expiratory muscles.

A.1.3 Other Inspiratory Muscles

The parasternals and the scalenes are considered the most important assessory inspiratory muscles. The parasternals run inferiorly and laterally from the lateral margin of the sternum to the 2^{nd} to 6^{th} ribs. Their action is to create the "bucket handle" movement of the ribs. The scalenes run inferiorly from the cervical vertebrae to the upper margin of the first two ribs. Due to the configuration of the first two ribs, contraction of the scalenes results in the "pump handle" movement . Other assessory inspiratory muscles are the sternocleiodomastoid , the levator costae and the serratus posterior superior.

A.1.4 The Abdominals

The abdominal muscles that have significant respiratory actions are the rectus abdominis, the external and internal oblique, and the transverse abdominis. The

rectus abdominis runs inferiorly from the sternum and the 5^{th} , 6^{th}, and 7^{th} costal cartilages to the pubis. The external oblique runs inferiorly and medially to the iliac crest. The internal oblique runs outwards from the inguinal ligament , the thoracolumbar fascia and the iliac crest to a range of attachments at the lower ribs and the rectus sheath. Finally, the transversus abdominis runs horizontally around the abdomen from the inner surface of the power six ribs, the iliac crest and the lumbar fascia to the rectus sheath and the linea alba (Osmond, 1995). The abdominals are primarily expiratory muscles. Their expiratory actions are: 1) to pull the abdominal wall inward and increase the abdominal pressure, causing the diaphragm to move cranially into the thoracic cavity and 2) to pull the ribs caudally and thereby deflating the lungs. However, the abdominals also assist in inspiration. During quiet breathing the abdominals play no role in respiration, but during exercise, as ventilatory demands increase, they contract rhythmically in phase with the inspiration. This action increases the abdominal pressure and assist the diaphragmatic action by resisting the outward displacement of the abdominal wall.

A.2 Structure and Morphology

The structural characteristics of the diaphragm and accessory muscles of respiration can be described in terms of the proportion of different fiber types present and the motor unit organisation of the muscle (Edwards & Faulkner, 1995). The adult diaphragm muscles are composed of 55 ± 5% type I, 21 ± 6% type IIA, and 24 ± 3% type IIB fibers (Lieberman *et al,* 1973). The intercostals muscles have 63 ± 2.7% type I fibers (Keens *et al,* 1978), with no significant difference between internal and external muscles. Although the percentage of type I fibers of the intercostal muscles is higher than that of the diaphragm muscle, the diaphragm demonstrates greater oxidative enzyme activity (Keens *et al,* 1978). The abdominal muscles are more variable in composition than the other accessory muscles with the range of type I fibers being 40-70%.

The diaphragm exhibits a twofold greater mitochondrial density than that found in limb muscle (Hoppeler *et al,* 1981). While the intercostals exhibit similar oxidative characteristics to the diaphragm, the accessory muscles show considerable variability in oxidative capacity.

Recruitment of motor units in the diaphragm appears to follow the general 'Henneman size principle' (Henneman & Olson, 1965). Most likely, motor unit recruitment in the diaphragm is similar to that of thenar muscles (thumb), where motor units reach 50% of maximum force at a stimulation frequency between 8 and 10Hz. According to Sieck (1988), recruitment by the size principle is to some extent moderated by the respiratory rhythm.

A.3 Function

The critical contractile variables in terms of understanding respiratory function are the maximum velocity of unloaded shortening (V_{max}), the force developed, and the ability to sustain force and power (Edwards & Faulkner, 1995). The time to peak tension and V_{max} of the respiratory muscles, being midway between fast and slow skeletal muscle fibers (Faulkner *et al,* 1979), are ideally suited to the diverse metabolic requirements of the respiratory muscles for sustained non-fatiguing contractions.

The respiratory muscles usually function at low afterloads, although increased resistance to airflow can raise it. As a consequence, the force-velocity relationship of fiber segments from the diaphragm muscle is intermediate between those of type I and type II skeletal muscles. Power decreases rapidly from the value produced during a single contraction to that sustained during repeated efforts. Following exhaustive exercise the sustained power may be reduced to approximately 30% of that generated during a single maximal contraction (Edwards & Faulkner, 1995).

The resting position of the diaphragm and to a lesser extent other respiratory muscles are in dynamic equilibrium. The elastic recoil forces of the lung, the effect of gravity on the ribs and the contents of the thorax all interact to determine this equipoise (Rochester, 1992). Contractions of the respiratory muscles, at various power outputs, act on the volume of the thorax in a manner that facilitates a diverse variety of ventilatory manoeuvres. The transformation of diaphragmatic muscle force to pressure may be estimated from the Laplace relationship, however, this is somewhat complicated by changes in the curvature of the muscle. Unfortunately, for other respiratory muscles, shortening length and force developed are impossible to measure. It is acceptable to estimate the external work performed by calculating the area of the pressure-volume curve (Edwards & Faulkner, 1995).

Contraction of the diaphragm forces the viscera downwards and thereby pushing the abdomen outward. The extent to which this lifts and expands the ribs is dependent on the abdominal pressure, which is determined primarily by abdominal resistance to displacement. The external intercostal muscles expand the rib cage with some assistance from the abdominals whose contraction during inspiration aid rib expansion by increasing abdominal pressure (Grimby *et al,* 1976). During normal breathing, although the diaphragm is consistently involved and exhibits the most active motor units, parts of the internal intercostals, and the external intercostals are active. Furthermore, the scalene and sternocleidomastoid muscles are frequently employed. Increases in minute ventilation are accompanied by progressive recruitment of motor units in each of the respiratory muscles, the exact pattern of which is dependant on the stimuli causing the hyperpnea (Derenne *et al,* 1978).

A.4 Summary

The structure and functional properties of the respiratory muscles reflect the unique functional requirements which they serve. The diaphragm in particular, with a high oxidative capacity, high maximum flow and an intermediate velocity of shortening is highly fatigue resistant. Furthermore, the abundance of highly oxidative fibers allows the diaphragm to perform at high percentages of maximum voluntary ventilation (Tenney & Reese, 1968) and as such is appropriately designed for the demands of exercise.

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